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THE PROGNOSIS OF INVOLUTION MELANCHOLIA*

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"Involution melancholia," sometimes spoken of simply as "melancholia," is one of the most frequent forms of mental disease, and the term is one of the oldest in psychiatry. Nevertheless, the clinical position of this group and the outcome of the psychosis are still matters of dispute. Until 1907 the disease was generally held to be a clinical entity with variable prognosis. For instance, in the seventh edition of his textbook, Kraepelin says: "It includes all pathological states of anxiety in more advanced age, which are not episodes in the course of other forms of insanity. Delusions belong also to this clinical picture in addition to the mood disturbances." As to the prognosis, he found that 32 per cent. of his cases were chronic, while 19 per cent. of his patients died within two years after the onset.

Only one important objection was made to the view that involution melancholia is a disease by itself. Thalbitzer [1] asserted that melancholia was related to the "angry mania" of Kraepelin and that the latter's third "mixed condition of manic-depressive insanity," the "depressive excitement," was practically identical with melancholia. These views, however, were not widely accepted.

In 1907 appeared a work by Dreyfus [2] which was rather revolutionary in its effects. He investigated the later development of eighty-

* This is a problem which interested the late Dr. Hoch for many years. In the spring of 1919 he began a review of 108 cases which he had examined between the years of 1895 and 1905 at the McLean Hospital in Waverly, Mass. Through the courtesy of Dr. Packard of the McLean Hospital and the superintendents of a number of other hospitals to which some of these patients had been transferred, it was possible to learn the outcome of the majority of the psychoses observed. Together we began an analysis of this material and reached some tentative conclusions. Since the untimely death of the originator of this work, a reexamination of the case histories seems to confirm these first formulations and justify a published report.—J. T. M.

1. Thalbitzer, S.: Melancholie u. Depression, Allg. Ztschr. f. Psychiat. **62:** 1905.

2. Die Melancholie, Jena, 1907.

five cases in which the diagnosis of melancholia had been made in the Heidelberg Clinic since 1892. He found that a large number of patients whose condition had been considered irrecoverable, even deteriorated, finally recovered, although the favorable outcome might not appear for almost ten years. The conclusions he drew have had much influence on psychiatric classification, for many physicians have followed him in regarding these cases as belonging to the manic-depressive group. He argued that many patients give a history of prior attacks and some pass from melancholia into a manic phase. This would indicate that the behavior of the psychosis is the same as that of the recognized forms of manic-depressive insanity. Patients fail to recover only in rare instances when the melancholia passes over into arteriosclerotic dementia. The ultimate prognosis is therefore as good as in manic-depressive insanity except for the frequency of death in these advanced ages. Finally, the clinical picture is made up of manic-depressive symptoms, particularly those of the circular "mixed conditions." Specificially these are, of the manic group: emotional variations (sometimes in the direction of euphoria but often as cessation of the depressive mood in the latter part of the day); excitability and irritability; need of communicating troubles; pressure of speech; flight of ideas; inflation of the ego; even with expansive ideas. On the depressive side the symptoms in common with melancholia are obvious: painful emotions of sadness, anxiety, etc., and inhibition of thought.

Both his evidence and arguments have carried much weight with psychiatrists, particularly in Germany. Kraepelin, for instance, has accepted them, practically in toto, and in the eighth edition of his textbook has eliminated melancholia as a separate nosologic category. One effect of this has been to make Kraepelinian depression a complex and puzzling picture. In it all kinds of clinical entities are assembled and many varieties of emotion, the one constant feature being an unpleasant affect, whether this be of sadness, anxiety or a painful tension.

Argument, however, may be directed against Dreyfus' assertions. The most crucial question is that of prognosis. Since his work psychiatrists have been more on their guard against making pessimistic prognostications, yet they know that some patients do not recover. For instance, in our series there are patients whose psychoses persisted practically unchanged for as long as twenty years without the development of symptoms pointing to arteriosclerosis. Invariability of recovery is, therefore, not a feature of involution melancholia. A history of prior attacks certainly tends to make one expect the psychosis in question to be benign, but we get the impression on reading his case reports that Dreyfus' zeal outran his judgment. In a number

of cases he ferreted out a history of depressions so mild as to seem to be neuroses or merely more or less normal mood swings. Variations of the emotional status are of great theoretic, psychologic importance, but they should not be called "psychoses" as long as their manifestations remain within certain limits. Otherwise nearly the whole world is, or has been, insane. As to his claims for the appearance of manic and other manic-depressive symptoms, we cannot find this convincing. There are, in psychiatry, few pathognomic symptoms. Clinical types are made up of certain groupings of symptoms. For instance, by selecting and putting together the proper features of a number of manic-depressive cases, one could easily paint a fair picture of dementia praecox. Yet no one would ally dementia praecox with manic-depressive insanity for that reason. Similarly, intensive study of a normal person may enable one to recognize in him all these symptoms. If sad, happy or worried, should he be called insane? Some of the symptoms he mentions are peculiarly nonspecific. For instance, emotional variations, excitability and irritability appear not only in normal reactions but in general paralysis, epilepsy, deliria, dementia praecox and manic-depressive anxiety. Other symptoms are extreme rarities in involution melancholia. Of these we may cite grandiose ideas (in pure form) and flight of ideas.

In spite of these objections, however, one feels that there is evidence for believing that some relationship with manic-depressive insanity exists. When a psychosis occurs that is one of a series in the same person, that is characterized by emotional disturbance and ends in recovery, it is hard to deny it kinship with manic-depressive insanity. On the other hand, if the chronic cases are to be included with the benign, this forces a modification of our ideas about manic-depressive insanity in an essential point. If the bar of good prognosis be let down, one would have to admit many cases of dementia praecox as well. An alternative is thinkable. It may be possible to differentiate between the benign and malignant melancholias, relating the former to manic-depressive insanity and the latter to dementia praecox. This could be done only if it were found that the prognostically bad symptoms were of a kind which we already view as schizophrenic in tendency. The results of our studies would seem to point in this direction.

Our program was to study the patients who recovered and thus determine what symptoms were benign. We then examined the histories of the patients who did not recover to discover the differences in their symptoms from those of the first group. Sufficient data were available in sixty-seven cases to justify their inclusion in this report. Of the others, it was either impossible to learn what the outcome of the psychosis was, or death ensued too early to justify any conclusion as to the chronicity of the disease. Among the patients who had

recovered it was found that the average period before improvement was nine and one half months; thirty-three improved during the first year of the psychosis, five during the second, one during the third and three during the fourth. One patient improved markedly after two years, then relapsed and began to recover only eight years after onset. The last was considered so anomalous a case as to justify the rule that patients with curable melancholias begin to improve in less than four years. We, therefore, have included among the chronic cases those of all patients who died more than four years after the psychosis began without showing any signs of recovery. Justification for this was evident when it appeared that the patients who died unimproved after this lapse of time showed the symptomatology of the chronic type.

Regardless of whether these cases shall be grouped with manic-depressive insanity or not, we believe it leads to greater clarity of understanding to discriminate between the typical manic-depressive depressions of early and middle life and the psychoses characterized as involution melancholias. We would define the former as reactions consisting of feelings of sadness, hopelessness, wickedness, incapacity and unreality on the subjective side, with objective evidence of physical and mental sluggishness and an appearance of dejection in attitude and facial expression; such delusions as appear are concerned with the moral obliquity of the patient, while hallucinations are extremely rare. In the melancholias, on the other hand, on a general background of such symptoms, appear fearful delusions and marked anxiety, often with terrifying hallucinations; a strong tendency to hypochondria sometimes leading to fantastic delusions; gross anomalies of conduct with much irritability, perversity and sometimes filthiness; finally, some patients show a loss of adequate emotional response to their environment or to their own ideas. Between these two types there are innumerable transitions, but between their extremes a marked contrast exists. (In the same way transitions are seen between manic-depressive insanity and dementia praecox.) What we will term the manic-depressive depression appears in later periods of life just as in youth and middle age. When there is no complication these patients recover and present no greater problem at this than at other ages. Our task lies with the cases of melancholia proper.

In his seventh edition, Kraepelin outlines the prognostic data of involution melancholia as follows: In general, an unfavorable outcome is to be expected with a loss of emotional reaction unaccompanied by a recession of delusions or with the development of absurd ideas. In milder cases, the emotional disturbance gradually disappears in company with the delusions, although the patients, in spite of an uncertain insight, become duller, their will power is weakened and they

become less active, often assuming an inferior or whining attitude. With greater deterioration delusions gradually fade away. The patients develop a poverty of ideas, their minds become unclear, and they become forgetful, indifferent and incapable of work. They have no insight but stand around dully, complaining monotonously. In some the definite picture of senile dementia develops. From this it will be seen that Kraepelin gives no criteria for predicting deterioration but rather describes what happens when deterioration takes place. We have attempted to study in retrospect the symptoms of the patients who recovered and of those who did not, in order to discover, if possible, what features in the earlier stages of the psychosis have a bad prognostic import.

SYMPTOMS OF INVOLUTION MELANCHOLIA

In order to present our material in concise form, the cases are arranged in tables showing the presence or absence of symptoms which we have come to regard as peculiar to involution melancholia and essential in the prognosis, while a supplementary table gives a synopsis of all or most of the symptoms presented by the same patients.

A brief description of these essential symptoms may make the tables clearer. The type of emotional reaction is briefly indicated so that no further explanation of this is needed. Different categories of delusions are given in the next three columns. The first category of death and poverty ideas is important because these fancies are highly typical of any kind of involution melancholia. Death is usually represented in a violent form—the patient is going to be hanged, electrocuted, cut up, burned, buried alive, and so on. The wild fear so characteristic of these cases apparently is often a response to these thoughts. Sometimes the fancies appear in disguise as a delusion of immortality. The patient cannot die, no matter how he may suffer from starvation, torture or injury. In other cases there may be an elaboration of the theme into thoughts of wholesale murder. All relatives, all friends, all nurses, patients and physicians have been killed or are going to be; dead bodies are piled around, and so on. Probably related psychologically to loss of life is the delusion of poverty. In its mildest forms this is merely a worry about expenses, in the most exaggerated types the patient is convinced that the family fortune is dissipated and his relatives are all in the poorhouse. None of the death or poverty delusions seems to have any bearing on the ultimate psychosis. Three quarters of all patients have them.

Hypochondria.—Even more frequent are the hypochondriac fancies. We have divided these into two groups which seem to have some fairly definite relationship with the outcome. In the first group, which we term "mild hypochondria," there are worries and complaints about health, pains, queer sensations, indigestion, constipation, etc., which are

identical with, or analogous to, the complaints every physician is constantly hearing from his nonpsychotic but neurotic patients. In involution melancholia, such imaginations simply tend to be more stressed. In the second type similar ideas are exaggerated into senseless and ridiculous delusions which often are associated with anal practices or other auto-erotic practices or fantasies. For instance, the patient declares he cannot swallow, although he may just have eaten; that his organs are all gone; that his bowels never move, that he passes so much by rectum that all the sewers are plugged; that an odor from him defiles the world; that he is paralyzed (while making movements); that he is only a foot high, and so on with endless variations. Such patients are often filthy in their habits, pulling feces out of the anus with their fingers, smearing the furniture and themselves. Frequently they masturbate shamelessly, pull ceaselessly at their lips, etc. One practice which might be considered auto-erotic but which occurs often in convalescing patients is that of picking at the fingers, which may be more of an expression of restlessness than of indirect onanism.

Irritability.—Some discrimination between the conduct of patients recovering and that of patients with chronic cases of melancholia may also be made. Although a certain degree of irritability is frequent in the more serious benign psychoses, there is a tendency to constant peevishness only when the prognosis is grave. These patients seem to resent any attention with rude words, scratching, biting and striking, sometimes with surly expressions of a desire to be left alone. This conduct is extraordinarily like that of a spoiled child in a tantrum.

Inadequacy of Emotional Reaction.—Deserving separate listing is the inadequacy of emotional reaction which occurs frequently in deteriorating cases. The psychoses with good prognosis show, as a rule, strong, even violent, affective symptoms. It seems to suggest a bad outlook when a patient has thoughts of being killed and is indifferent. A more serious sign is a narrowing of the mental horizon when interests one by one disappear and attention is focused more and more on the patient's body or on his troubles. These patients wander around monotonously whining a stereotyped complaint. A small but rather definite subgroup is that group of patients who have what we have been accustomed to term organic insufficiency, since the whole picture seems to reflect a fundamental and general senescence rather than a localized cerebral abiotrophy with senile dementia or a largely psychogenic disturbance, such as characterizes so many of the involution melancholias. These cases usually begin with insomnia followed by a gradual loss of interest. If there are any self-accusations or paranoid or death ideas, they are in the background of the clinical picture. There are hypochondriac ideas, but these usually are con-

cerned with the patient's condition in a vague, general way. Specific complaints are usually about constipation, with slight exaggeration, but they are never absurd or ridiculous. The patients usually wander aimlessly around, whining, unoccupied and apathetic. Sometimes there is mild restlessness, not accompanied by poor sleep, however, as in the benign cases. Organic insufficiency seems to have an invariably bad prognosis.

BENIGN CASES OF INVOLUTION MELANCHOLIA

Examination of Table 1 reveals some interesting data. Both death and poverty ideas and mild hypochondriac notions occur often, not infrequently dominating the clinical picture. In only one case (Case 26) were these fearful ideas expressed without anxiety, but this patient, on the other hand, sometimes showed affect. Some mention of severe hypochondria occurs eight times, but in four of these cases the remarks thus catalogued were occasional. Of these, three patients made such complaints only during a short episode, two of them showing at this period a special agitation. In one case (Case 4) the remarks were apparently similes rather than delusions, while in two more (Cases 27 and 43) it seems on reviewing the records that the patients may have been using comparisons rather than expressing convictions. In Case 8 the hypochondiac notions were sometimes delusions and sometimes only metaphorical. This patient and the patient in Case 43 were the only ones who frequently expressed these ideas and neither had a consistent and obvious belief in the fancied disability. In no case do we find well developed severe hypochondria to be a dominating, permanent symptom, so that we can assert that benign cases fail to show this common feature of involution melancholia prominently or consistently. The only patient (Case 8) who gave frequent utterance to unquestioned delusions of this nature was at the menopause. This deserves notice, as will appear later.

As to the behavior which we list under the heading of peevishness, only seven of the forty-three patients who had recovered showed symptoms of this nature, and in most of them it was confined to mere irritability; in only one (Case 43) was this more than an occasional phenomenon. It is interesting that only one in this series had a longer psychosis than this patient.

To summarize the material presented in Table 1, these patients showed marked emotional reaction, usually anxiety with restlessness, and they had prominent delusions of death and poverty. Peevishness and severe hypochondria were isolated symptoms which were never sufficiently well developed to be consistently dominant features in the psychosis. Patients began to recover after an average duration of nine

TABLE 1.—Recovered Cases

No.	Sex	Age	Emotional State	Death or Poverty Ideas	Mild Hypochondria	Severe Hypochondria	Peevishness	Improvement, Years	Recovery, Years	Varia
1	♀	47	Moaning, restless	++	+	.....	.....	7/12	1	
2	♂	46	Retardation	.....	.....	.....	.....	1/12	1/2	Typical manic depressive
3	♀	66	Retardation	.....	.....	.....	.....	3/12	5/12	Typical manic depressive
4	♀	64	Complaining	++	+	As similes only	.....	1/2	3	
5	♂	68	Retardation	.....	+	.....	.....	5/12	9/12	Typical manic depressive
6	♀	59	Retardation, then anxiety	++	.....	.....	.....	3 3/4	4 1/4	Mixed manic depressive and involution
7	♂	56	Agitated anxiety	+++	.....	.....	.....	4/12	8/12	
8	♀	48	Retardation, anxiety, fidgety	+++	.....	++ often only similes	+	2/12	4/12	Menopause
9	♀	51	Retardation with spells of elation, sometimes apprehensive	+++	.....	.....	.....	1 +	1 +	
10	♂	45	Dulness and anxiety	+	+	.....	.....	5/12	6/12	
11	♂	66	Retardation, restlessness	.....	+	.....	.....	1 5/12	1 6/12	Manic depressive type
12	♀	61	Agitation and despair	+	.....	.....	.....	10/12	1	Manic depressive type
13	♀	46	Apprehension, restlessness with some restraint	+	+	.....	.....	6/12	8/12	
14	♂	47	Retardation, inadequacy, self-blame	.....	+	.....	.....	5/12	8/12	Typical manic depressive
15	♀	56	Apprehensive, restless, reticent	+	.....	.....	.....	2/12	1	
16	♀	45	Inadequacy, guilt, apprehensive, episodic agitation	+	.....	.....	.....	5/12	9/12	
17	♀	55	Fear, agitation	+++	.....	+	.....	10/12	1	Severe hypochondria, episodic
18	♀	49	Fearful, agitated	+	+	.....	.....	2/12	3	
19	♂	68	Dejected, listless, episode of agitation	+	.....	+	.....	1/5	1/3	Severe hypochondria, occasional during brief period of agitation
20	♀	54	Languid, complaining, agitation with hallucinatory episodes	+	+++	.....	.....	1 9/12	4	Recovered after manic episode
21	♂	63	Retarded, dejected, at times surly	+	++	.....	+	3/12	6/12	Manic depressive type
22	♀♂	45	Varying retardation and apprehension	+	+	.....	.....	3 1/2	4	
23	♂	64	Hopeless, worrying, episode of agitation	+++	+	.....	.....	1	1 1/2	

24	♂	56	Restless, fretting	++	++	.....	.....	5/12	6/12	
25	♂♀	51	Retardation, negativistic	.....	.....	.....	+	4/12	5/12	
26	♀	59	Aprehensive or calm	+++	+	.....	.....	4/12	3 1/2	Fearful ideas often expressed without affect
27	♂	61	Sad and hopeless, then apprehensive	+++	+++	.....	.....	6/12	7/12	
28	♀	45	Crying, hopeless, inactive	.....	+++	Idea that sex organs were shriveled up, may have been a simile	.....	2	2 5/12	Menopause
29	♂	66	Hopeless, inadequacy, worry	.....	++	.....	.....	4/12	6/12	
30	♀	50	Variable depression with worry and compulsive thoughts	.....	+	.....	.....	Constant variation	5	
31	♀	46	Retarded, episode of mild stupor, depressive worrying	+	+	.....	+	6/12	3	Content mainly self-accusations
32	♀	39	Distress with many somatic complaints	+	+++	.....	+	5/12	7/12	
33	♀	52	Retardation, discouraged, a little restlessness	.....	+	.....	.....	7/12	6	
34	♂	49	Periods of agitation, otherwise whining lassitude and feeling of inferiority	++	++	+	+	1 9/12	2 3/12	Severe hypochondria only once during a spell of agitation
35	♂	46	Discouraged, inadequacy	+	+	.....	.....	4/12	1 4/12	Manic depressive type
36	♀	42	Sad, gloomy, crying, slight restlessness	+++	.....	.....	.....	5/12	6/12	
37	♀	58	Gloomy, worrying	++	+++	.....	.....	1 2/12	1 4/12	Two subsequent attacks with recovery
38	♂	54	Some inadequacy, feeling of sin and worry	++	+	.....	.....	7/12	1 3/12	
39	♀	50	Listless, retarded, occasional queer smile, at times fear	+	.....	.....	.....	8/12	10/12	
40	♀	47	Mild retardation	.....	.....	.....	.....	5/12	1 7/12	Typical manic depressive
41	♀	51	Retardation with some restlessness	+	.....	+ transient	.....	6/12	7/12	
42	♀	50	Crying depression, inadequacy	.....	+++	.....	.....	3 8/12	4 3/12	
43	♀	48	Great restlessness with moaning and vague distress	+	+++	??	++	11/12	5	Frequent complaint that she could not swallow, may not have been meant literally

+ = Occasional.
++ = Frequent.
+++ = Dominating the clinical picture.
Manic depressive = Ordinary depression characterized by retardation with feelings of sadness, wickedness, inadequacy and unreality.

TABLE 2.—Chronic Cases

No.	Sex	Age	Emotional State	Death or Poverty Ideas	Mild Hypochondria	Severe Hypochondria or Autoerotism	Peevishness	Restriction of Interest or Affect	Improvement, Years	Chronic, Years	Varia
44	♀	63	Extreme irritability; mumbles to self; complains of fear	++	++	.....	+++	.....	12, following hemiplegia	14 Died	
45	♀	52	Tremendous agitation; later seclusive, apathetic, with irritable periods	.....	.....	+++	++	.....	7 1/2	8	
46	♂	68	Unoccupied, insufficient affect.....	+	+++	.....	.....	Narrowing of mental horizon with shallow affect	.....	12 Died	
47	♀	66	Restless, moaning, marked agitation, wants to be left alone	+++	.....	+++	.....	Usually insufficient affect	.....	6 3/12 Died	
48	♀	56	Restless, moaning, distressed; episodes of great agitation	++	.....	+++	++	.....	.....	8	Constant hallucinations of hearing
49	♂	64	Great retardation with sadness and feeling of unreality	.....	.....	.....	.....	.....	2 10/12	7 Died	Typical manic-depressive but arteriosclerotic
50	♀	50	Constant restless fussing, peevishness and demand for attention	.....	+++	+++	+++	.....	.....	18 6/12	
51	♀	59	Sometimes restless, constant stereotyped moaning and affectless complaints	+	+	.....	.....	Narrowing of mental horizon with shallow affect	.....	11	
52	♀	43	At first retardation; later restless, apprehensive complaining; finally spells of irritability	+	.....	+++	++	.....	5	20	Prominent ideas of diminution
53	♀	60	Restless, distressed moaning, very uncommunicative	+++	.....	+++	+++	.....	3?	18 Died	
54	♀	48	Monotonous complaints, with shallow affect, except when delusions are questioned, then irritable	++	+	+++	+++	Shallow affect as a rule	.....	5 Died	
55	♀	68	Three stages: (1) restless, hopeless, vague apprehension; (2) agitation one week; (3) chronic peevishness	+++ (for one week)	+	+	+++	.....	.....	6 Died	
56	♀	59	First, depressed; later, restless with shallow affect	+++	.....	+++	+++	.....	.....	11 7/12 Died	
57	♀	54	Inadequacy onset; restless and vague fear; finally chronic moaning and complaining	++	+	+++	.....	Narrowing of mental horizon	.....	4 3/12 Died	
58	♂	56	(1) Worry and suspicion; (2) mild retardation; (3) chronic suspicion	+++	.....	.....	++	.....	.....	11 Died	Constant hallucinations and delusions of dementia praecox type
59	♂	54	Unoccupied, surly, seclusive.....	.....	+	+++	+++	.....	.....	4 2/12 deteriorated	Marked change of character 9 years before psychosis
60	♂	57	Worry, at times restless, at times subdued, chronic uneasy muttering	++	.....	+++	.....	.....	.....	9 6/12 Died	
61	♀	52	Restless, complaining, peevish.....	++	+	+++	+++	.....	.....	5 Died	
62	♂	60	Listless, slow but insufficient affect....	+++	.....	+++	+	Insufficient affect	.....	12 Died	
63	♀	52	Great restlessness, hair pulling, etc., with vague apprehension; later no affect, still restless	.....	.....	++	+++ with deterioration	Gradual loss of affect with narrowing of mental horizon	.....	19	

and one half months, while recovery occurred from four months to six years after onset with an average total duration of twenty and one half months.

CHRONIC CASES OF INVOLUTION MELANCHOLIA

Table 2 shows a marked contrast in symptomatology. The cases recorded in this table are of those who did not recover during many years, the duration of the psychosis up to the last report being noted in the "Chronic Years" column. Included also are the cases of patients who died without having shown any sign of recovery within four years after the onset or who seemed to have reached a chronic state some years later. As has been explained, all those who recovered showed improvement within this period, so that one is justified in assuming that death terminated a chronic psychosis.

In general, the emotional states or behavior of the patients with malignant melancholia are different from those of patients with benign cases. Instead of frank fear reactions, there were much moaning, whining, seclusiveness, surliness and often insufficient affect. Restriction of interest or affect occurred in ten out of twenty cases. In each of these patients this loss was permanent, not subject to variations as in the patient in Case 26 who recovered, and it was always a prominent feature of the clinical picture.

About the same proportion of chronic and recovering cases show death or poverty ideas, and these are of the same type roughly, so that probably these ideas have no significance for prognosis.

A marked contrast is evident in the incidence of hypochondria, however. Roughly, three fourths of the patients with benign cases showed this symptom, but in only one sixth was it a dominant characteristic. Of the chronic cases, on the other hand, eighteen out of twenty made such complaints and of the two who did not, one had a case of pure manic-depressive depression complicated by arteriosclerosis (Case 49[3]), so that one may say that hypochondria is almost always present in the more serious cases. It tends also to be more pronounced in its form, for in fourteen cases of the twenty it was a dominant symptom. The same tendency to exaggeration of interest in the body is evident in the frequency of severe hypochondria (an occasional symptom in the recovering patients) and of actual auto-

3. This patient showed some improvement nearly three years after the onset of his psychosis and might, of course, have eventually recovered had he lived. But since this improvement did not continue during the next four years, this seems unlikely. The case is included because it probably is one type of chronic psychosis—a benign reaction in which recovery is prevented by purely physical factors.

TABLE 3.—Doubtful Cases

No.	Sex	Age	Emotional State	Death or Poverty Ideas	Mild Hypochondria	Severe Hypochondria or Autoerotism	Peevishness	Outcome	Varia
64	♀	53	Great restlessness.....	+	+	+++	+	Observed 14 months; discharged to another hospital; returned home "much improved" 3 2/12 years after onset	Menopause
65	♀	69	Retardation, then apprehension, then elation, then retardation and anxiety	+++	.....	.....	.....	Improved after 2 years; then relapsed; 8 years after onset improved enough to be taken home from another hospital; 6 months later sister reported her well	Sexual ideas suggestive of dementia praecox
66	♀	49	Dejected, peevish.....	+	+	.....	++	Observed for 6 months, then taken home; 4 years later husband reported she was well except for resentment at having been put in hospital	
67	♀	62	Great restlessness, with self-accusations; later, quieter with same ideas but uncommunicative	.....	.....	Constant analerotism	.....	First improvement noted 2 years and 8 months after onset; well enough to go home 7 years after onset; not heard of since	

erotic practices, fifteen, or three quarters of the patients, manifesting this preoccupation; in thirteen of these it was a dominant symptom.

Another minor symptom in recovering patients which is prominent in this second group is the exaggeration of irritability which we speak of as peevishness; fourteen of the twenty showed it, and in these it was a dominant feature.

It is evident that the three symptoms of attention to the body, peevishness and restriction of interest or affect are definitely associated with a bad prognosis. Not one of these occurs as a persistent or dominant symptom in the recovering melancholias, while with two exceptions every patient with a fatal or chronic case showed one, two or all three of these malignant symptoms in marked form. The two exceptions rather go to prove this rule, for neither showed a typical picture of involution melancholia, one being a manic-depressive depression with arteriosclerosis and the other very much like the picture of a dementia praecox with constant delusions and hallucinations.

DOUBTFUL CASES OF INVOLUTION MELANCHOLIA

If these two tables included all our material, dogmatic statements might be allowable. Unfortunately, however, simplicity and rigidity of definition are rarely justified in psychiatry and this study of involution melancholia offers no exception. In Table 3 appear data which seem to render doubtful the finality of the foregoing conclusions. Four cases are represented here which were not placed in the first two tables because the question of complete recovery was settled merely by report from relatives. We would have been justified in eliminating these from the series, were it not that three of them might have been expected to develop chronic psychoses according to the foregoing criteria. Whether these patients recovered fully or not cannot be demonstrated, but this, for our present purposes, is not so important. They certainly improved enough to return home. Had they been thoroughly examined by a competent psychiatrist and found to have still some psychotic tendencies, this would mean little. Mild mental abnormalities are so frequent in old age as to be almost universal, so that our standard of normality, high for youth and middle age, must be lowered in considering cases of the senium. On purely theoretic grounds we might say that nearly all old people have symptoms of involution melancholia, with their worries about health and property of themselves and of their families, but practically, we are dealing with exaggerations of these tendencies which proceed to such development that the unfortunate ones become incompetent and intolerable burdens in the home. If then, a patient becomes well enough to return home and stay there, the presumption is that the accentuation of senile traits—the psychosis in this narrow sense—has disappeared. Let us consider these cases one by one.

The patient in Case 64 showed no insufficiency of affect, but there were constant complaints of the type which we have come to regard as malignant. She thought her rectum was broken off, that the urine came out of the wrong place, that nothing could pass to her stomach, etc. In addition she was occasionally peevish. She was, however, at the menopause. If one examines the two climacteric cases in Table 1, it is seen that these patients tend symptomatically toward the malignant type. For instance, the patient in Case 8 frequently indulged in extreme hypochondriac delusions and was sometimes peevish. The patient in Case 28 constantly complained that her sexual organs were shriveled up. These three patients, being at the menopause, constitute a small group, the members of which one might have expected to have chronic psychoses, who recovered or improved markedly. Although this group is small, the presence of demonstrable physical factors probably allows us to regard them as different from the usual patients with involution melancholia. It is not difficult to imagine that the endocrine disturbances of this period may facilitate a graver degree of mental disintegration than is possible without deterioration in normal physical health.

Case 65 is a complicated one, in which the patient had mood variations like those of a younger manic-depressive patient. She had many sexual delusions reminiscent of dementia praecox. She thought she had been made pregnant by her brother-in-law, that men visited her room at night; she also complained of vaginal sensations. Had she been 30 or 40 years younger one would not have hesitated to make a diagnosis of dementia praecox. It was not until she had been ill for eight and one-half years that she began to improve and was taken home. We have only her sister's word to support the belief in her recovery. It may well be that—at the age of 77—a senile or arteriosclerotic dementia wiped out the delusional and emotional symptoms. At any rate this patient's clinical picture did not coincide with that of involution melancholia, so that we are probably justified in disregarding it in our general conclusions.

Case 66 was that of a patient who was frequently peevish but who showed no other unusual or malignant symptoms. Her case was not included in Table 1 chiefly because the dates of her improvement and recovery were not known. Her continued resentment at having been placed in a hospital may have been merely indicative of a character change rather than of an unrecovered psychosis in the narrow sense.

Case 67 presents real difficulties. Self-accusations dominated the clinical picture with tremendous restlessness—a mixture, as it were, of manic-depressive depression and involution melancholia. For a long period she continued to smear feces over her clothing and even to pull them out of her rectum with her fingers. Later she became quiet and uncommunicative. By all our standards this patient should have remained in a state of deterioration, and it was with surprise that we learned of her improvement in a state hospital to which she was transferred, and of her removal to her home. No argument can explain this case. It remains, quite frankly, an exception. But in any series of sixty-seven psychiatric cases, is not at least one exception to general rules encountered?

COMPARISON OF DREYFUS' FINDINGS WITH THOSE OF AUTHORS

One is naturally interested in comparing the foregoing findings with the observations of Dreyfus. This is a difficult matter because

data such as the exact nature of false ideas are not carefully noted in his case histories in many instances. Further, it must be remembered that his patients were under treatment at a time when melancholia was generally thought to be frequently a chronic disease, so that patients not showing early improvement were regarded as chronic and quite possibly observed superficially and at long intervals. This may account for the statements that the patients in Case 13 and Case 20 did not show signs of improvement until after eight and six years, respectively. In general, his material seems to follow the rules which we have deduced from the series here reported. The possible discrepancies alone deserve mention.

In Case 3 the patient complained for a month of being filled with feces and of being unable to defecate. Whether these ideas were qualified does not appear. At the end of the month doubt was admitted as to the validity of these complaints. We would probably have listed this case as showing an episode of bad hypochondria. The patient in Case 6 during a period of four months often smeared feces. One suspects, however, that the clinical picture was more like that of manic-depressive insanity than that of involution melancholia. No false ideas are reported; for one day, at least, elation was present. Smearing is, of course, a frequent symptom with manic patients, particularly with those who become absorbed in their unexpressed thoughts.

In his group of fatal cases no patient with fantastic hypochondriac delusions had recovered before death.

Most of the patients in his "not yet recovered" group were obviously on the way to normality.

One patient (Case 32), who apparently had the benign type of melancholia, developed arteriosclerotic dementia and had, therefore, a chronic case like that of the patient in our Case 49. In Case 31, although there were complications, the patient showed no definitely malignant symptoms and apparently recovered. Apparently she did not leave the hospital because she had become "institutionalized" as so many old people do, and was apprehensive of quitting that comfortable environment. The patient in Case 30 had only during the onset the delusion that her stomach was closed and that nothing could pass through it. After many variations in her clinical picture she began to recover three and one half years after onset and was apparently improving steadily when reported. This case we would classify as showing merely an episode of severe hypochondria. Case 29 is interesting. The patient showed for a year, while under observation, persistent and dominating hypochondriac delusions. She was then taken home. Eight years after the onset she was reexamined. She was still hypochondriac, was careless of herself and her home, did not go out, and had a feeling of insufficiency. This picture looks like that of chronic deterioration, yet Dreyfus calls it manic depressive insanity with hysterical tendencies!

In general, then, a review of Dreyfus' material gives us no urgent reason to doubt the validity of our conclusions.

A word should be said about the nosologic position of involution melancholia. This analysis of symptoms tends to throw the material into two groups. In one, with a good prognosis, there is as a rule, a strong affective reaction and the delusions are of the same order as those appearing in manic-depressive insanity. In the other, the patients deteriorate and show symptoms distinctly of the kind we associate with dementia praecox; the affect is apt to be insufficient, the behavior is auto-erotic and negativistic in the extreme, and the delusions are ridiculous, expressing perverse sexuality. Plainly, then, involution melancholia belongs in two markedly opposed psychiatric divisions. There are two ways of meeting this difficulty. It may be regarded as a separate division with a variable prognosis or as two related psychoses. In the latter case one would regard the benign melancholic psychoses as a type of manic-depressive reaction (like mania, depression, stupor, etc.) and the deteriorating psychoses as a type of dementia praecox. It is probable that individual taste is likely to determine the classification adopted by psychiatrists, at least for many years.

An argument in favor of looking on involution melancholia as a reaction type of manic-depressive insanity is found in the clinical pictures of many manic-depressive patients. Conditions of marked fear of death, of loss of property, or of moral destruction, with hypochondria but accompanied by no real sadness or retardation are common as episodes or as entire psychoses in persons of early as well as advanced years. If space permitted we could, for instance, detail the history of a patient, aged 33, whose case caused much difference of opinion as to diagnosis. He recovered completely. On reviewing the full and accurate notes of the case it appears that symptom for symptom the clinical picture was a duplicate of that of involution melancholia. Had he been twenty or thirty years older there would probably have been no confusion of opinion among those who observed him.

It is not within the scope of this report to discuss the psychology of involution melancholia. It may be remarked, however, that the regressive mechanisms of the malignant psychoses seem closely related to those of dementia praecox, while the analogy holds just as strongly between the psychology of benign cases and that of manic-depressive insanity.

One point should be mentioned of theoretic and, possibly, great practical importance. In both Dreyfus' and our own material it seems a rare event for a melancholic patient to recover fully while still in the hospital. The return to complete normality occurs at home in the patient's normal environment. This is in marked contrast to the phenomena of recovery in most cases of manic-depressive insanity, with the exception, perhaps, of the stupors.

SUMMARY

We may state the following as the result of our work: The results in a series of sixty-seven patients (summarized in Table 4, pp. 18-37) in whom the final outcome was determined in all but one case, cause us to conclude that patients with involution melancholia recover unless they show as dominant symptoms: marked insufficiency of affect, peevish or auto-erotic behavior, or ridiculous hypochondriac delusions which usually are concerned with the alimentary tract. These prognostically bad symptoms may be present for a short phase of the psychosis in women at the menopause without their prejudicing the outlook for recovery. All patients who eventually recover show some improvement within four years after the onset. The others run a chronic course or die unimproved.

TABLE 4.—CASES—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation	Severe Hypochondria
1	47	0	Restless, moaning	Suicidal attempt	People plot against her	.....	.....	.....	.....
			Restless	.....	Will be taken away	.....	.....	.....	.....
2	46	1	Gloomy, unoccupied; sometimes restless; marked inadequacy	.....	Worries over inadequacy; will not get well; what will become of family?	Blames self for condition	.....	.....	.....
3	66	4+*	Unoccupied; inadequacy with slight restlessness	.....	Nothing to live for; fears losing mind; worry over condition	.....	.....	.....	.....
4	64	0	Unoccupied; fussy manner and complaining	I will be dead tomorrow	.....	.....	.....	No cars running; no fire, no water, no clothes	No room for food; no stomach; does not urinate; all as similes, not actual delusions
5	68	1	Inadequacy; dejected	.....	Will not recover; worry over condition; wife will come to want	.....	.....	.....	.....
6	59	0	Inadequate for her work	.....	Losing her mind	Killed husband by her jealousy; not religious enough; will be taken to court	.....	.....	.....
			Depressed two weeks	.....	Will not get well				
			Sometimes restlessness; worry, jealous of husband and friend, ashamed of it; "mixed up," "I change my mind so often"	Will be killed, hanged, for killing husband	Husband dead				
			Much distress	Will be buried alive; will be put in dark place, death tonight and terrible punishment, ideas of reference in connection with death; putting flowers in water means will be drowned, etc.	Home broken up, husband has remarried; in prison	Terrible thoughts of people, poisoning, etc.; feels responsible for them	.....	.....	.....
7	56	2	Agitation, wringing hands, tense, anxious, suspicious (tastes food); indefinite dread	Fears poison	Will go to ruin; everybody against him; indefinite dread of something to happen	Has not done business well	.....	.....	.....
			Two weeks: more agitated, shouting, breaking glass, throwing furniture	Nurses going to put him under ground, going to burn him, going to murder him					
			Quieter, still suspicious and having feeling of dread						
8	48	0	Inadequacy, retardation, some fear, fidgety	Place in hell for her	.....	.....	Real world? real people? suspended in space; everything elongated and enlarged	.....	No stomach, no mouth, no head, "shrunk up," no opening to body, does not breathe through nose; (only similes when criticized)
9	51	1	Depressed; retarded; exhilarated spells	Fear of burning, dogs will eat her, may be hanged or dissected	.....	.....	.....	.....	.....

* Manic circular.

—Studied by Authors

Mild Hypochondria	Analerotism	Autoerotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
.....	.....	.....	.....	Husband untrue; later he accuses her of infidelity	Will be poor	.....	Begins with menopause
Epigastric uneasiness	.....	Picking fingers	.....	.....	Idea of poverty		
....	.....	.....	.....	.....	Worry over expenses	Poor sleep when restless	
Pressure in the head	No passage down her throat; nothing passes her; both as similes	Picking fingers	.....	.....	No money to stay here; no money to buy clothes	Marked insomnia	
Burning sensation and pains through limbs	.....	.....	Complains about conditions on ward	.....	.....	Poor	
....	.....	.....	.....	.....	.....	.....	Sister's throat is cut
.....	....	.....	.....	.....	.....	.....	Attacks of indigestion; heightened blood pressure also some months after discharge
.....	.....	.....	Spat food out of mouth, knocked dish out of nurse's hand; quarrel over trifles	.....	No money, no clothes, poverty, scared about luxuriousness; refuses food because cannot pay; later because no stomach		
.....	.....	.....	.....	.....	.....	Mild sleeplessness	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation	Severe Hypochondria
10	45	0	Rather dull, not retarded; apprehensive	.....	Something terrible might happen: people waiting outside; will be arrested	Thought "somebody might have led me astray"	.....	.....	.....
11	66	2	Marked inadequacy, with some restlessness	.....	.....	Never amounted to anything, did not take enough interest; degenerate; never had a decent trait	No affection	.....	.....
12	61	1	In despair, colossal restlessness, moaning, rocking	Something is going to destroy her; markedly suicidal	Going to lose her mind; frightful thoughts	"I have wrecked their lives, killed them all, shall lose my mind and it is my fault"	"Can't sympathize, can't enjoy"	.....	.....
13	46	0	Worried, restless, apprehensive	Fears will be tortured	Something dreadful will happen	.....	.....	.....	.....
			Restlessness, with immobile face, certain constraint, slow in answering questions referring to her worry	Will be cut up	Afraid will be punished; police waiting; will be harmed	Disgraced sister; said she stole from employer; then "not guilty," never did any harm			
14	47	3	Depressed, dull, inadequacy	.....	No hope, no salvation	Defaulted money; no salvation, people know his real character	.....	.....	.....
15	56	1	Restless, hard to say what she worries about, "mixed up"	"Are they going to kill me?"	Fears poison and reading of her letters; worry about home and children	Is wicked	.....	.....	.....
16	45	2	Inadequacy, guilt, episodic agitation	Suicidal	Family branded for life; prison; something will happen to husband; arrested; police spies about	.....	.....	.....	.....
17	55	3	Depressed, listless	Going to die	Dreadful calamity	.....	"Sometimes think I am not alive, I have so little feeling for others"	.....	Contagious disease; would infect everybody by unclean breath; depopulate cities; daughter lost position and husband hanged on account of odor; stomach does not work, a hollow there
			Often marked restlessness and moaning	Will be thrown on dump, naked, torn to pieces, head cut off, eyes dug out, murdered, tortured; husband torn up	Poison in food; vitriol in wash water				
18	49	0	Marked restlessness, whining, wailing, especially in spells, looking very distressed; complaints of being "all discouraged"	.....	Feeling as if world were going to fall on her; mind going, "will be like my father" (had melancholia)	.....	.....	.....	.....
19	68	1	Dejected	Onset: will die, last day on earth	Onset: arrested; house will fall down	Has done much harm, dishonest	.....	Has destroyed the world; no life	For five days: substance all gone, stomach full of corruption. This with agitation, broken sentences and confusion

—BY AUTHORS—(*Continued*)

Mild Hypochondria	Analerotism	Autoerotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
.....	.....	.....	.....	.....	"Money gone"	Good sleep	
Pins and ants in extremities; crawling feeling all over, throbbing feeling in chest, fullness in abdomen	.....	.....	.....	.....	.....	Some sleeplessness	
Agony sensations in head	.....	.....	.....	.....	.....	Fair as a rule; occasionally very little sleep	
A terrible strain about the heart	.....	.....	.....	.....	In poverty, will be put on street	Poor sleep at first only	
Pinched feeling in head							
.....	.....	Picking and biting nails	.....	.....	No money; will starve		
Distress in head							
Indigestion; stomach burns	Food is urine; the tube by which fed is rectal tube	.....	.....	.....	.....	Insomnia	Severe hypochondria, merely episodic
Peculiar aches in legs and hands, with a desire to move, also "emotional suffering feeling"	.....	.....	.....	.....	Money going		
.....	.....	.....	.....	.....	.....	Good sleep	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation	Severe Hypochondria
20	54	0	Languid, tired	.....	Mental powers gone; won't get better; sister insane, will be blind, will be electrocuted for killing somebody; completely mad in a way never heard of—like a mad dog	Why was I born? Am I to blame?	.....	....	Burning up; worms crawling over her
			Sudden marked restlessness, distress, even fear, suffering, excitement	Devil said he would murder her; she will be electrocuted					
			Quiet, marked depression, crying occasionally; gradually hopeful but anxiety about sister						
21	63	4	Retarded; sometimes restless, often surly	Gang waiting to kill him (once)	Things going wrong at home; has been ruined by syphilis	.....	.....	....	.....
22	45	0	Marked restlessness with unequal retardation	.....	Will be tried; something will happen to husband and children	Killed husband, children and sister	.....	....	.....
23	64	1	Lethargy, worry, depression, restlessness, hopeless of recovery	.....	Never get well, what will happen to family?	.....	.....	....	.....
			Depression, but occupation and better evenings						
			Two days stirred up, reticent, suspicious, began at night suddenly when he got restless "to be kept for life"	Will be used for vivisection	Will be tortured, will be experimented on; yet says himself ideas are wild, though not losing suspiciousness				
			Improvement 1 month, though still gloomy without special ideas	.....	Never get well				
			Depressed, languid, indifferent but can smile	.....	Apprehensive but not without insight; will be left on street; at police station; it will kill his family, etc.				
24	56	1	Restless, moderate fretting over trifles	.....	Afraid of becoming insane	.....	.....	....	.....
25	51	4	Retardation, negativistic (probably result of suspiciousness)	.....	.....	.....	.....	....	.....
26	59	0	Worried, restless, anxious	Will be cremated, crushed to death	Put in dark hole, will be burned, put in lake of fire; wild animals tear her to pieces; also delusional interpretations of things heard	.....	.....	....	.....
			Sometimes composed, speaking of apprehensive ideas, often guilt, anxious and appealing to physician						
			After three weeks, following visit of husband, sudden improvement, henceforth variations						
27	61	1	Depressed, hopeless, then apprehensive	Will be killed, burned, drowned, strangled	Will never recover	Ought to be an outcast	.....	....	.....
28	45	0	Depressed, cried; said had no ambition	.....	.....	.....	No interest, no affection for family; does not care about appearance; sleep unnatural; ability to appreciate is dulled; pain sensations and touch different; no natural feeling in head	....	Sex organs shriveled up (simile?)
			Unoccupied, lack of energy, especially in forenoon, better evenings, much bitter crying						
29	66	0	Typical inadequacy feeling of awful depression; worry	.....	.....	Life a failure; neglectful of duties	.....	....	.....

Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
Burning skin, pain in back, sore muscles, nerves tingling, "all gone" in stomach, mind weak	.....	.....	.....	.....	.....	Fair sleep	"Worms crawling over her," probably a death idea
Pain in abdomen, gas in bowels, nose dry, does not sleep	.....	.....	Often surly	.....	Family extravagant	"Does not sleep"	
.....	.....	.....	.....	Onset: calls herself whore because she exposed herself in tearing clothes; stayed in bed and attracted men			
Prickly sensation in body, head, knees and forearm	.....	.....	.....	.....	Property to be taken to defray expenses of keeping him in asylum and family will be destitute; money going; when all gone will be turned in street	At first fair, later excellent sleep	
Epigastric sensation; disease of lungs	.....	.....	.....	.....	Family are paupers, daughter going to poorhouse	Considerable insomnia	
.....	.....	Picking of fingers	Frequently negativistic				
Disagreeable sensation in feet and hands	.....	.....	.....	.....	.....	Poor sleep	
Syphilis of throat (had syphilis in youth)	.....	.....	.....	.....	Poorhouse		
Empty feeling in stomach as if food did not reach bottom; loss of sexual feeling and of all sensations	.....	.....	.....	"Sexual organs shriveled up"; attributes this to coitus interruptus. Sex feeling gone, petrified clitoris also accounts for lack of energy	.....	Usually quite poor sleep	Dry skin and lack of perspiration in hot bath
Dyspepsia, cannot sleep, smothering sensation	.....	.....	.....	.....	.....	"Can't sleep"	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
30	50	4	Constant variations; quiet, some inadequacy, slightly depressed; worry; compulsive thoughts	.....	.....	Innumerable trivialities	Things do not look quite natural; cannot cry; feels as if turned into stone	.....
31	46	2	Much retardation at first, also stupor symptoms, gradually freer, sometimes somewhat restless; agitated with self-accusations	Ought to die, cannot die	Has to pay for everything	(When freer) the vilest wretch, has dragged family into poverty, has terrible disease	.....	.....
32	29	0	Gradual neglect of work and appearance; attacks of restlessness with marked sensations, writhing, pinching herself, pulling mouth out of shape, crying for help, "do something" Intense restlessness, gradually diminishing with great distress; constant sensations	Onset: Asks to be killed; fears suffocation; is afraid will be put into a box	Is afraid will have a fit, a stroke or go insane	.....	.....	Family all dead
33	52	1	Moderate retardation, sometimes slight restlessness with "suffering" Very melancholy without content but great taedium vitae	.....	She and daughter will be punished	Misappropriated money	.....	.....
34	49	0	Depressed, gradual improvement	Attempted suicide	Never get well			
			Sudden agitation, cannot swallow food, everybody dead, etc. Unoccupied, whining, discomfort, distress, and weak apprehension, much lassitude and feeling of inferiority	Will never die, will be left on earth alone (as punishment for misdeeds); voices say he will be taken into dark, broken on stones; thought he would be boiled when some one said "lobster"	Will be put on a paralytic's water bed; people here have their teeth knocked out Human bodies ground up downstairs; this with strangeness of things made him think buildings were made of suffering human bodies; also that food and wicker chairs were flesh; ideas of reference	"Why am I so wicked? I don't want to do such a dreadful thing"	(Retrospective) everything seemed unreal; there seemed general dissolution; every thing changed—room bigger	Everybody dead; no newspapers; no countries; fields not worked, seasons too short for crops to ripen; time shorter; no cars running; all business stopped; wife not real (because he thought she was dead); no one eats or sleeps
35	46	0*	Quiet, inadequacy, discouraged	.....	Everything looks bad; borrows trouble	Ought to be ashamed of himself	.....	.....
36	42	0	Dread constantly complained of, especially marked at night; moderate restlessness shown in picking, some crying; sad gloomy appearance	Fears she will die before night; food poisoned; going to be cut up; four men will kill her	Fears something may happen to husband or son; poison in food; to be taken away at night; her teeth will be extracted; all to be burned; husband to be killed; wind signifies this; toast passed in queer way means she wants to kill husband with toast; tea brought to her while husband is there means she is going to kill him and it is to brace her up, etc.	Cross stitches mean to keep the cross before her; odd things others say are to impersonate her so that she is blamed	.....	.....
37	38	0	Gloomy, worrying	.....	.....	.....	.....	.....

* Depressive makeup.

Intense Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
.....	Epigastric uneasiness	....	.....	.....	Sexual feeling when another (female) patient touched her breasts			
.....	Has terrible disease	....	Wets and soils	Resistive to treatment	.....	Dragged family into poverty	.....	Pregnant at entrance
.....	Throat full as if contracted, as though some one were grasping it; as though eyes were coming out of socket, as though tongue was pulled down, palate swollen; numbness and pricking in jaw and throat (inside); feels as though hollow way down through (outside); numbness all over as though dying; prickly all over; stomach discomfort; head: fullness, big as a drum, something boiling in it all the time; band sensation, numb	....	.....	Struck, scratched and kicked nurses	.....	.....	Sleep disturbed at first	Improved as she understood sensations had not significance she ascribed to them
.....	Throat filled up; stomach growing up	....	.....	.....	.....	.....	.....	Everybody knows her thoughts because things happen when she thinks of them
Cannot swallow, anus "stopped up" (complaints occurred only during one spell of agitation)	Pains all over body; lets himself fall, says he is too weak to get up; afraid of falling and breaking in pieces	....	.....	Often desires not to be disturbed; begs to be left in his room; whining with skewed up face; often has to be dressed	.....	Worry over money	Poor sleep	
.....	Head heavy	....	.....	.....	.....	His people will come to want	Sleep, good	
.....	For year fussing over stomach trouble, idea of cancer or heart disease	....	.....	.....	.....	.....	Poor sleep	
.....	Constant fear of going blind, also thought was growing deaf or paralyzed	....	.....	.....	.....	Cannot pay bills	Irregular	Two subsequent attacks with recovery

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation	Severe Hypochondria
38	54	0	Mild restlessness, some inadequacy, feeling of sin and worry	No right on earth	Soul is lost, has committed unforgivable sin, is in devil's power	Sinner, unpardonable sin, lost self-control, touched his wife's genitals	Spirit taken out; does not worry enough about family	.....	.....
39	50	1	Listless, retardation, occasional queer smiles; sometimes fear	Speaks of leaving the world soon	Something dreadful going to happen; microbes on her	Contaminates people with microbes	.....	.....	.....
40	47	2	Mild retardation	.....	.....	Very wicked but cannot be made to say what she worries about	.....	.....	.....
41	51	0	Restless with inadequacy	Going to die	Afraid will be taken away	.....	.....	.....	Transient during onset: throat closing up
42	50	2	Depressed, inadequacy, somewhat agitated, crying	.....	Will never get well; mind failing; teeth decaying; hair falling out	.....	.....	.....	.....
43	48	2	Restless, moaning, marked agitation, will not answer questions, wants to be left alone	.....	Something dreadful to happen; hopeless condition; asks to be protected	.....	.....	.....	Throat comes together so that cannot swallow
44	63	0	Dull and restless	Will be blown up with dynamite	.....	.....	.....	.....	.....
			Extreme irritability; sometimes complaints of fear	"Come and cut my head off"	Put on street, never see daughter again; dominated by fear of being left alone	.....	.....	.....	.....
45	52	0	Worry, then outbreaks of agitation	.....	.....	.....	.....	No one about her, is a dressed up form; a wilderness; no daughter, no husband; (orientation?) "I don't know anything about it"	No heart, mouth, stomach, pulse
			Tremendous agitation, with aimless acts, running about; later secluse with periods of apathy and irritability						
46	68	0	Depressed, agitated spells, again natural	Suicidal tendency	Soul lost	.....	.....	.....	.....
			Dull depression; feeling of going to pieces; apathy	Can't die	Apprehensive ideas about harm to daughter and son-in-law				
47	66	0	Worry over things said and expenses	.....	Police coming, people waiting to take her; food poisoned	.....	.....	No water about, no home, "no anything"	Odor from her flesh; cannot pass feces or urine; bowels coming out; urine is more blood than urine
			Restless, moaning, afraid in spells, otherwise quiet; moaning, worried, with insufficient affect		Carriage to take her to funeral or wedding				
					House falling to pieces				

—BY AUTHORS—(*Continued*)

Mild Hypochondria	Analerotism	Autoerotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
Precordial sensations	.....	.....	.....	.....	Will lose everything and go to poorhouse	Good sleep	Ideas of reference; e. g., spoons rap when he does not eat well; attributed to higher power and to others
Heart disease; afraid of cancer; again pregnant	.....	.....	.....	Claims assault and pregnancy; again cancer	.....	Poor at first only	
Teeth decaying; hair falling out	.....	.....	Fussing about eating	.....	.....	Usually good	
Choking sensation in throat, cannot swallow, can hardly breathe	.....	.....	Peevish, complaint about no treatment, wants to be left alone	....	Worry over financial affairs		
. .....	.....	.....	.....	.....	All property lost		
Bad feeling all over, throat dry, stomach on fire; feels full and stopped up	.....	.....	Peevishness with dissatisfaction, irritability; hit nurses, threw furniture; surly expression, swore, very obscene	.....	.....	Poor sleep	Spelling words backward and constant mumbling; improved after a stroke
....	(After three years) may urinate anywhere	.....	Assaultive at times; always irritable; often resented treatment	.....	.....	Sleep, sometimes good, sometimes bad	
Lack of nutrition owing to poor teeth; semen oozing all the time from him	Stomach full, crowded by bowels (no physical basis)	.....	.....	.....	Patients took his money, will be destitute	Insomnia	Constant narrowing of mental horizon and insufficiency of affect
.....	Soiled, refused to go to closet, "bowels don't move," "they are coming out"	Marked tendency to strip	.....	Fear of men coming into room to assault her. Letter sent to say she is a bad woman	Worry over expenses; bills can't be paid; thousands expended on her. No clothes, no home	Poor sleep	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
48	56	0	Inactivity; depression Restless; distressed Spells of blind excitement, with intense distress and fear (sex content) Moaning, frightened, still sexual, also hypochondriac; intention paralysis	Can never be killed Will be killed or burned	.....	.....	Body different; everything gone except seeing and knowing Patient says to her "recede," "elongate," and then she shrinks up; complains of periods when everything looks queer	.....
49	64	7	Played out; restless Quiet, retarded, immobile, has to be dressed, etc.	.....	.....	Worst man in world	Everything is wrong, nothing is right	.....
50	50	1	Worry over condition Marked restlessness, fussed up, constant demands for attention	.....	Harm coming to husband and self	.....	.....	.....
51	59	0	Easily fatigued, worry. Standing, walking restlessly, moaning much; marked narrowing of mental horizon; affect seems sterotyped and shallow	Cut up	Heating apparatus will explode; prison; ruined; exposed on street in glass case; terrible thing to happen; dear ones in the cold, etc.	.....	.....	.....
52	43	1	Depressed, retarded; moderate restlessness; suicidal, almost mute, with decided restlessness Moderate restlessness with freer speech; apprehensive; finally spells of irritability	Cannot die, (repeated attempts at suicide); asks if he will be hanged	"I am afraid"	Wicked, responsible for all wickedness in world; made all my people sin, lost my soul	"No life in me, seems as if my limbs were dead"; body different; throat not right; things do not look right, are larger	.....
53	60	0	Distressed, dejected, moaning, restless, some perplexity	Can't live or die	"Afraid of everything," feeling of responsibility about everything	"Have lived on false pretenses"	.....	.....
54	48	1?	Nervous, crying, then "I changed in one night when all my power was taken away"; much depressed, in utter despair; later shallow affect except when delusions questioned, then irritable	Tried suicide; can't die; asks to be cut up and put in bottle	Will never get well, no use to anybody; legs numb and paralyzed, dead	"God changed me and I don't know why"	Shriveled up, face drawn up, mouth shrinking; entirely dissolved, only an image, dead; only a ball of wire, only a little ball, grown blind; "I think only when I speak and when I stop speaking there is not a thought in my head"	.....

—BY AUTHORS—(*Continued*)

Intense Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
Stomach gone; is only a shell; can't walk; will be blind; can never get killed; body different, no bones; can't breathe; everything gone except seeing and knowing; shrinks up	Head feels hardened, feels as if she could not breathe on account of blocking in throat	Incontinence during limp spells	Strips at bidding of voices	Kicked, bit, struck in episodes associated with great excitement; later struggled against tube feeding, then relaxed and urinated on floor	Hears man, hypnotized, sexual commands, feels vaginal sensations; frequent stripping and coming into hall naked	.....	Insomnia	Constant hallucinations of voices
.....	Head not right, "sad all over, have not been made right"	.....	.....	.....	.....	.....	Good sleep	Arterio-sclerotic
Can't hear or see; is dead, (occasionally) hands stiff; can't breathe through nose	Pain, discomforts, hands stiff, heavy feeling in neck; will have cancer	Bowel trouble	Much shameless masturbation and occasional wetting; talks to self, "Why don't you get out of the window dearie?" "Don't you want to see how you look?"	Constant fault-finding; sometimes aggressive, violent to nurses with irritation, constant demands for attention	Exposes herself			
... .	Leprosy	.....	.....	.....	.....	Ideas of poverty	Irregular	
Body different, throat not right, tongue too large	.....	Untidy	.....	Spells of irritability				
She has only two hands and feet; flesh not right, no body, getting smaller and smaller, drawing up, various sensations (touching face) make her smaller, "everything looks big"								
Rotten through and through, mouth gone, tongue swallowed, neck broken, throat broken by feeding, looks very old, feet are dead and he can't walk	.....	Wilful bed wetting	.....	Sometimes resistance and distinct desire to be left alone; pronounced muscular negativism; more and more nothing could be obtained from her	.....	No money, a pauper	Insomnia at times	
Stomach and bowels grown up, brain turned to bone; organs of abdomen crowding in chest, entirely dissolved; only a ball of wire, a rag doll, a rat, a bit of lip, etc.	Legs numb and paralyzed	.....	Pulls at lip until it is deformed	Highly irritable when ideas are questioned	.....	.....	Insomnia at onset	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
55	68	*	Depression, restless, worry over condition	.....	"I shall go to pieces, I feel so badly"	.....	.....	.....
			Restless movements, looks worried, craving sympathy, moaning					
			One week of intense agitation, not even time to eat properly, shouting "murder"	Will be burned, tortured, furnace ready, cremated, can read it in faces about him	.....	.....	.....	.....
			Only slightly restless but very fault finding; no ideas	.....	Will not get well but owing to bad treatment			
56	59	0	Depressed, inactive	.....	"I am crazy; I will kill myself"	Reproach for not having cared better for son	.....	.....
			Gradual restlessness but shallow affect with delusions of violent death	Will be poisoned; will be thrown out of window; cannot die	Will be put in dungeon with crazy man; "You are going to crush the life out of me," will be thrown out of window, into a cellar, heavy chains rattle which will be put on her	"They say I have killed my boy by giving him strychnin, but I did not do it, the doctor did it"	.....	.....
57	54	1	Onset with inadequacy, then restless writhing; gradually more monotonous affect and narrowed interests	Will be suffocated, buried alive, isolated	.....	.....	.....	.....
58	56	1	Makeup: apprehensive about health; since 14 nervous dyspepsia, often thought he was going to die; for twenty years had to have wife constantly with him even on business trips; hypersensitive and retiring	.....	.....	.....	.....	.....
			Worry, anxious	Suicidal ideas, going to be killed tonight; thought he was dying because his hands felt numb; eggnog makes him feel numb, and he does not want to die without his senses	Indefinite dread of something to happen; thought incorrectly written check scheme to injure him and family; conspiracy, spies	.....	.....	"My friends are destroyed. The world destroyed. Voices say the world was taken out of its axis; there will never be another world"
			Two weeks: languid, slow action, never constant; moderate restlessness, moaning, decided suicidal tendency, some suspiciousness. One year: unoccupied, restless, then settled down; sometimes perplexed expression; suspicious watching, and tendency to withhold information, feeling of mental restriction and that he does not understand what things mean		Afraid to take bath; after refusal thinks friends are sacrificed in his stead. Friends being killed; they don't get home; ditches in garden are made for them; wife dead; all non-Catholics destroyed			
			Then chronic hallucinations and delusions with insufficient affect and perfect orientation	Many ideas about self and others being killed, world destroyed, etc.	Feeling of significance which he does not understand. "There is a clearly definite movement and I am the only one who does not understand its significance or when to move." His actions prescribed; his life surrounded; occurrences have meaning; appliances and machinery destroying friends and world; mind reading and double thinking. "Why do you keep putting men from South Africa, South America and South United States into holes in the ground? Why do you take them to the attic and shoot them down the chutes?"	.....	.....	.....

* Many for ten years.

—BY AUTHORS—(*Continued*)

Severe Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
..... Feet are mortifying	Throbbing sensation in head	For years afraid of soiled things, ashes and dust, and afraid she might step in something that was not right	....	.....	"It will kill my brother when he hears what the cause of my disease is, The people will think my disease is of a private nature. I brought the disease on myself; law says, 'Thou shalt not lust' "	.....	Always insomnia	
.....	.....	.....	....	Very fault finding, peevish, irritable; treatment, food, etc., bad				
.....	.....	.....	....	.....	.....	.....	Usually very poor	
Bones and neck broken; throat dead; odor from it; dead inside because cats and crows are after her; bowels ruptured fifteen years ago, mortification stuff spreads from them, air is full of it; this has caused a railroad accident and death of her boy, is making every one crazy; other patients are mortifying; has hydrophobia; is an animal	.....	.....	....	Peevish, chafing, petulant at times, irritable to nurses yelling at them when prevented in her restlessness from disturbing them				
Can't eat, can't swallow; food does not go to stomach	Will be blind; things blurred before her eyes	.....	....	.....	.....	.....	Marked insomnia	
.....	.....	.....	....	.....	.....	.....	Irregular, usually poor	
....	Head does not work well	.....	....	At times very interfering and troublesome	. .....	"Have I been stripped of my property?"	.....	Expansive ideas; voices say "He ruled the world and all the people in it. He settled the world." When he eats they say "He eats a king, a queen, a prince." When he took something out of a candy box, "He is eating his children"
....	. .....	.....	....	.....	.....	.....	.....	Hallucinations and delusions of dementia praecox type

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
59	64	3	Curled up in room; surly expression, certain restlessness, totally unoccupied, untidy	.....	Afraid of harm; conspiracy to give him "pox"	.....	.....	"They say this is a hospital"
60	57	0	Worry, ruined self by taking poison, uneasy, restless, will be taken to court, family, etc., will be indicted Quiet, subdued Restlessness Restlessness more marked; "O God, I have to get out of here" Restless, much worried; had to get out; everything troubled him but could not describe it; finally chronic muttering	Attempted suicide (oxalic acid); going to be hanged	Held here for criminal trial "will astonish everybody." Something with post office All gone, "O God, O God, the whole business is gone, it is awful, awful, all gone to pieces"	.....	.....	.....
61	52	0	Nervous, hypochondriac Marked restlessness, talking incessantly, repeating words and sentences; gradually more exclusively interested in hypochondriac ideas, with irritability when questioned	Will be buried alive	Will be tried for crime of which she feels innocent; refused food at first because covered with poison, later because organs are gone	.....	.....	.....
62	60	2	Depressed about business Inactive, gloomy, listless, slow in speech and act, "I have no life, no force" No restlessness, same listlessness and inactivity, pauses in speech, decidedly apprehensive ideas but accepts these hopelessly and resignedly; insufficient affect throughout	Will be killed in various horrible ways	Ruined fiancée's life; certain forebodings not crystallized "They are coming to take me away." Heard team drive up in night, knew family in it, dead, and they would get him when they came back (hallucinations); going to be killed, "and then voices came and called my name." Will be burned in furnace, new one to be built because old one filled with ashes of dead men, "enough men for a month." Cut up in a machine, killed by electricity, thrown down a hopper lined with knives; conspiracy to destroy him and friends and others; mentions two men who he thinks have already been killed; gases collecting inside him and when they accumulate he will explode	"It seems as though all my life the mainspring of my actions was not right." Has been selfish all his life but was not so bad as people thought	.....	.....

—BY AUTHORS—(*Continued*)

Severe Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
Is rotten, odor from him smells up everything	Has "pox"	Filthy	Much masturbation	Objects to everything, sneering manner at examination; surly expression	.....	.....	Poor sleep at first	
.....	Pain in head	Smears, will fill toilet or pipes if he goes to water closet (worry of expense stopped when this came); had diarrhea and tried to defecate all over place so as not to fill up pipes; complained often of being covered with feces; throughout tendency to defecate and urinate on floor	Rubs head or buttocks	.....	.....	The bills are not paid; they must be running up into the thousands; did not eat on account of expense	Insomnia	Always poor performance with intellectual test
Organs gone, therefore can't eat; heart stopped beating, all bones broken; only 16 years old; only 3 feet high; no heart; no organs, they passed out in her stools and filled the cesspool; bowels broken; burning sensation all over body which makes her strip constantly, attributed to poison put in bed by nurses	Burning sensation all over; worried about heart disease	Will not go to closet; digs out her feces	Strips constantly	Whining, petulant tone, with much irritability; can also be made to smile at times; complains of this awful treatment (poison put in her bed by nurses)	.....	.....	Fair sleep	
No room for food, gases collect inside; further accumulation will make him explode; disagreeable gases come from him	No life; no force	.....	.....	Refusal of food	.....	Lost all property; has tangled up estate entrusted to him and will never be able to settle it; hospital people will take all his property away	Good	

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
63	52	0	Lost interest, restless, more so when husband goes away; duller, in bed; ideas	.....	Will be arrested, "Don't take me down there"	Wicked, had not treated husband right, to be arrested; inadequately formulated guilt; did wrong in coming here, being here, etc. "Don't think I am insane, I am not." "I was thinking I would tell you why I came and what I did to my brother-in-law and sister." "I was going to tell you of my coming here, I ought to have told you, I knew I was doing wrong but I kept on staying."	.....	.....
			Marked restlessness, pulling out of hair or motions of hands, considerable distress; unchanging expression, at times puzzled (exophthalmic goiter); little spontaneous talk, slowness in answers; later free. "My thoughts are not on it" to questions; finally practically no affect and stereotyped talk about going home or being in hospital			"I want to confess, my people are innocent and I am guilty (repeatedly), can't seem to tell what I worry about; it seems gone from me now; I have put it off so long that now I can't tell. I have forgotten."		
64	53	0	Restlessness, so that feet get swollen	"If I can live without eating, can't die, can't even be killed"	Something may happen to son	.....	Hands feel different, no feeling in them; entirely different from any one else	.....
65	69	0	Languid, sad	.....	.....	.....	.....	.....
			Retardation, shame (sexual), and mildly apprehensive	Will be killed, brother will be killed; she will be put into dark hole	House will tumble, last meal	Is bad	.....	.....
			Apprehensive	Same	.....	.....	.....	.....
			Exhilaration, volubility, slight flight	None	None	.....	.....	.....
			Well behaved					
			Gradual return of apprehensive ideas, restless, moaning and depressed	Will be killed	Will be tortured; she and family will be put into black hole	Has done dreadful things	.....	.....
66	49	0	Gloomy depression, peevish	Onset: will be hanged; later, "Let me die." or "Why don't you chop me up?"	Electricity, poison in food. "I suppose I have to go to jail." Ideas of reference about objects	Projected; accused of being hypocrite, thief, prostitute, living with man not her husband	Nothing natural, everything strange; husband not husband; she not Mrs. H., does not know who she is, doctor and hospital not doctor and hospital, nurses are paper girls; "I thought we had a real war"	.....
						Wicked		

Severe Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
.....	.....	Occasionally soils when mentally deteriorated	.....	Resistive with final deterioration	.....	.....	Sleep almost normal in spite of diurnal restlessness	Short episode of exophthalmic goiter
Examination of uterus caused mortification of spine; something snapped; body broken in two; rectum broken off; gone around the waist; will be blind; throat stopped up; nothing passes to stomach; can't be burned to ashes, would just roast	.....	Rectum broken off; urine comes out wrong place	.....	Whining, peevish	.....	.....	Often poor sleep	Loses flesh
.....	Poor health	.....	.....	.....	.....	.....	Poor sleep	
.....	.....	.....	.....	.....	Brother-in-law at instigation of sister had made her pregnant Every forenoon says somebody had visited her at night Vaginal sensations			
.....	.....	.....	.....	.....	Some one in room at night	.....	.....	Drops sexual ideas with greater anxiety
.....	.....	.....	.....	.....	None			
.....	.....	.....	.....	.....	Men come in at night; pregnant; vaginal sensations			
.....	Malaise, pain, vomited; is given medicine to keep bowels from moving; somebody has spoiled her body	Food is offal (as a metaphor)	.....	Disinclined to answer, "Please go away." More and more reticent, "said too much already." Wants to be left alone, pulls away petulantly when touched; irritability with striking and tearing things	Did not put curtain down when took bath. "If different men came every night, I did not know it." "It was shown to me that I practiced self-abuse"	No money to buy anything	Poor sleep at first only	Reported well four years after onset but could not forgive husband for taking her to hospital

TABLE 4.—CASES STUDIED—

Case No.	Age	Previous Attacks	Emotional State	Death Ideas	Other Apprehensive Ideas	Self-Accusation	Unreality	Negation
67	62	0	Uneasiness, fretful Restless, cannot sit down; intensely worried; voluble; cannot give data of life "because constantly thinking of wicked deeds done" More restless, temporarily quite intense; striking head against wall, crawling into fireplace, putting hand to throat as if to choke herself Restlessness marked, also marked curiosity, going around house looking into other people's rooms, reading their letters, etc.	Attempted suicide	.....	Sin; is the evil one; spoke of remorse of conscience; niece and boarder did not get along, should have separated them, greatest crime in history, such wicked deeds. "When I was a child we played we were killing each other, then we acted angry and later I could not help doing the same and feeling the same, and now it has gone on until it has come to this." Not insane, has killed many people by pretending to be (predominant theme)	.....	.....

—BY AUTHORS—(*Continued*)

Severe Hypochondria	Mild Hypochondria	Anal-erotism	Auto-erotism	Irritability, Peevishness	Adult Sexuality	Poverty Ideas	Sleep	Varia
.....	.....	Constant putting of fingers in rectum and smearing feces; asked why did so, "Must have something to do"	.....	.....	.....	Her clothes all stolen	Poor sleep	

SPINAL SUBARACHNOID BLOCK AS DETERMINED BY COMBINED CISTERN AND LUMBAR PUNCTURE

WITH SPECIAL REFERENCE TO THE EARLY DIAGNOSIS OF CORD TUMOR

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For some years the spinal fluid with the so-called Froin syndrome has been considered almost pathognomonic of compression of the spinal cord. This opinion has recently been reiterated by Leschke [1] and by Lantuéjoul,[2] each of whom collected a series of cases, the one chiefly from German sources, the other from the French. The practical value of the recognition of this fact is, however, almost nil because such fluids are usually found only after destruction of the cord, too late for operation or other therapeutic procedure.

For a much shorter time—about a decade—has it been known that a spinal fluid apparently normal in every respect except for protein increase is frequently to be found below a cord tumor. But fluid presenting isolated protein increase could hardly be considered more than suggestive of compression of the cord because a similar picture could be otherwise obtained. Nevertheless, in cases presenting the clinical picture of cord tumor a fluid containing marked increase in protein, and not otherwise abnormal, is a valuable correlative aid in diagnosis because it speaks for a degree of compression which is not necessarily destructive. If we could only be sure that such a fluid indicated tumor in a given case, it would be a valuable sign. Such evidence is furnished by a method published by me last year,[3] by means of which it is possible to demonstrate not only that the lumbar fluid is pathologic, but that the fluid above the tumor is different, and, most important of all, that an obstruction exists between the two.

At the time of writing the previous paper only nine patients had been examined by the combined cistern-lumbar technic; it is now possible to report on seventy-one such examinations in sixty-five patients.

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1. Leschke: Ueber die Gelbfarbung (Xanthochromie) der Zerebrospinalflussigkeit, Deutsch. med. Wchnschr. April 7, 1921, p. 376.

2. Lantuéjoul: La Coagulation Massive et Spontanée du Liquide Cephalo-Rachidien, Rev. Neurol. **36**:339 (April) 1920.

3. Ayer, James B.: Puncture of the Cisterna Magna, Arch. Neurol. & Psychiat. **4**:529 (Nov.) 1920.

Method

The method is simple and easy to comprehend, but must be carried out with care. It must not be forgotten that while cistern puncture is easy it is potentially dangerous, and is absolutely contraindicated in certain cases of increased intracranial pressure.

The patient is placed on one side so that the points of puncture, cistern and lumbar, shall be on the same horizontal plane. A glass tube with water and an air-bubble to serve as a level has proved helpful in accomplishing this. After local injection with procain puncture of the lumbar sac and of the cisterna magna are made in turn (the technic of the latter is fully described in two papers previously published [4]) and each needle is fitted with a three-way stopcock, to the upright arm of which is attached a glass manometer. The fluids are then allowed to run into their respective manometers and readings made. Oscillations with pulse and respiration and changes of pressure on coughing and deep breathing are noted in each tube, but especially important is the increase in pressure on sudden accession of cerebral venous pressure. This may be easily produced by gentle pressure on both jugular veins in the neck. There is reason to believe that all of these oscillatory phenomena and pressure changes are normally equal in the two manometers. The stopcock of the lumbar needle is now opened and fluid released into a graduate, noting whether or not the cistern fluid falls during the procedure, and the same is repeated for the cistern needle noting the pressure in the lumbar manometer. The pressure in either manometer should fall promptly, usually 30 to 40 mm. following release of 5 c.c. of fluid from the other needle. After enough fluid from each locus has been withdrawn for examination it is well to repeat the observations on pulse oscillations and the effect of jugular compression. The needles are then withdrawn. The whole procedure should be accomplished in about fifteen minutes, with no more inconvenience to the patient than the discomfort of two punctures and the retention of a somewhat strained position for the length of time required.

The fluids are examined in the usual manner for cells, Wassermann and colloidal gold reactions, but especially for total protein, for which a quantitative method is desirable.[5]

In order to insure comparable readings in the two loci, it is necessary that each set of needles, stopcocks and manometer tubes be alike. The bore of the latter should be about 1.5 mm. to give the best visible oscillations and needles of 18 gage have proved most satisfactory. It is essential that the punctures be cleanly made, as blood-tinged fluids will vitiate both manometric and protein readings.

NORMAL FINDINGS

As we were not justified in performing combined punctures on normal persons, and as the method is obviously applicable only to the living, we have been forced to draw our conclusions as to normal from a variety of evidence. For example, a number of patients under treat-

4. Wegeforth, Ayer and Essick: The Method of Obtaining Cerebrospinal Fluid by Puncture of the Cisterna Magna (Cistern Puncture) Am. J. M. Sc. **157**:789, 1919. Also footnote 3.

5. Denis and Ayer: A Method for the Quantitative Determination of Protein in Cerebrospinal Fluid, Arch. Int. Med. **26**:436 (Oct.) 1920.

ment for optic atrophy and general paresis by means of serum administered by the cisterna magna route have afforded opportunity for double puncture; also numerous patients have been investigated by this method who were thought to have degenerative cord lesions, but in whom the possibility of cord tumor was raised, and a few of these patients having normal fluids have been examined surgically with negative findings, and one has come to necropsy, in whom no block had been demonstrated. Most convincing of all were the findings in two patients examined before and after removal of cord tumors, in whom the preoperative and postoperative findings were fundamentally different. A consideration of all of these leads us to conclude that normally the spinal subarachnoid space permits free passage of fluid in either direction, and that the composition of cistern and lumbar fluids is almost identical.

If we are correct in our assumption that normally the spinal subarachnoid space affords perfectly free communication to fluid, it is obvious that with two manometers, one at each end, we may demonstrate this continuity by lowering or by raising pressure at either end and reading the effect in the manometers. Various means for changing the pressure have been employed: Withdrawal of fluid and deep inspiration lower pressure; coughing, crying, grunting and injection of fluid raise it. Compression of both jugular veins, leading as it must, to increased cerebral venous pressure, has been found a reliable method of raising pressure and withdrawal of fluid the most satisfactory method of reducing it. Moreover, in using these two means there is no danger from the possible introduction of harmful substances into the meninges. All of these changes appear promptly and to an equal degree in both manometers in cases in which we believe the subarachnoid space is unobstructed. One more observation has been found to be of value in the manometric study of the spinal fluid; the pulse oscillations should normally appear in both manometers, but in cases apparently normal it frequently occurs that the oscillations in the cisternal manometer are greater in amplitude than in the lumbar, suggesting the cerebral rather than the spinal arteries as the source of both.

It will be seen that all of these observations constitute in reality a dynamic study of the fluid circulation within the spinal subarachnoid space. The demonstration of continuity or obstruction by these hydrodynamic tests forms the most reliable criterion on which we base our judgment of spinal subarachnoid continuity or of block.

Combined cistern and lumbar puncture affords us also an opportunity to examine both fluids in the laboratory. We know what normal fluid from the lumbar sac should be, and in this series are a number of cases in which all tests—chemical, cytologic and biologic—are almost

identical in the two loci, and coincide with our idea of normal. We must believe that normally the two fluids are nearly the same, although a slightly greater protein content in the lumbar fluid appears to be constant. While the two fluids are frequently similar, a slight or a moderately greater protein reaction is obtained in fluid from the lumbar needle. The exact amount of normal difference in protein content of the two fluids is as yet debatable, and as it is the protein alone that has been repeatedly shown to be increased below cord tumors, this normal limit is of the utmost importance.

In that protein increase below obstruction has been repeatedly shown by numerous observers to be of transudative origin, we may consider these tests as primarily designed to show stasis.

It is, therefore, reasonable to conclude that under normal conditions hydrodynamic tests show the spinal subarachnoid space to be freely open, and examination of the fluids from the lumbar sac and cisterna magna shows them to be similar in character.

The findings in this series of sixty-five cases may be grouped as follows:

1. Those which we regard as normal. These have just been considered.

2. Those showing subarachnoid block, as demonstrated by hydrodynamic study, and also by increase of protein below the block (hydrostasis).

3. Those showing no block by dynamic tests but with well-marked difference in protein in the upper and lower fluids.

This paper deals especially with the second group in which evidence of block is clearly demonstrable.

CASES PRESENTING HYDRODYNAMIC AND HYDROSTATIC EVIDENCE OF BLOCK

In eighteen of the sixty-five patients examined by this method block was demonstrated according to both criteria. In all except one a satisfactory explanation was found, eleven by operation, three by the roentgen ray, two by a history of previous meningitis, and one by the effect of antisyphilitic therapy. Table 1 gives the data of chief importance in this correlation, with regard to these eighteen patients. Points of interest are: (1) that the block was shown between the ages of 13 and 59 years; (2) that all levels of the spinal cord are represented in the table; (3) that block was demonstrable whether caused by intramedullary, meningeal or extradural agents, in which (4) many pathologic types are represented. Of greatest importance is the fact that of the eighteen patients, nine showed clear, colorless fluids below the block as well as above it, abnormal fluids the significance of which might well have been overlooked on casual examination.

TABLE 1.—PATIENTS IN WHOM BLOCK WAS DEMONSTRATED BOTH BY HYDRODYNAMIC AND HYDROSTATIC TESTS

Name	Age	Duration of Symptoms of Cord Compression	Appearance of Fluid		Pathologic Findings			Determined by	Remarks
			Cistern	Lumbar	Level	Location	Agent		
Mr. C.	38 years	6 months	Colorless	Colorless	Thoracic	Intramedullary	Gumma	Operation	Case 2
Mrs. C.	33 years	15 months	Colorless	Faint yellow, no clot	Thoracic	Intramedullary	Glioma	Operation	
Mr. F.	37 years	2 weeks	Colorless	Deep yellow, no clot	Lumbar	Extradural	Dislocated vertebra	Operation	Illustration 1920 report†
Mr. G.	24 years	4 months	Colorless	Faint yellow, no clot	Thoracic	Meningeal	Cholesteatomatous cyst	Operation	Case 3, 1920 report†
Mr. H.	41 years	7 years	Colorless	Lemon, no clot	Cervical	Meningeal	Chronic syphilitic meningitis	Operation	
Miss B.	13 years	10 weeks	*	Lemon, no clot	Cervical	Intramedullary	Glioma	Operation	
Mr. R.	13 years	3 weeks	Colorless	Colorless	Cervical	Extradural	Enchondroma	Operation	Case 4
Mr. S.	30 years	3 months	Colorless	Colorless	Thoracic	Extradural	Tuberculosis of spine	Operation	
Dr. A.	43 years	5 months	Colorless	Colorless	Thoracic	Extradural	Perineurial fibroma	Operation	Case 1
Mr. D.	42 years	18 months	Colorless	Colorless	Cervical	Intradural	Perineurial fibroma	Operation	
Mrs. J.	59 years	3 years	Colorless	Lemon, no clot	Thoracic	Extradural	Chondrosarcoma of spine	Operation	
Mr. S.	28 years	6 weeks	Pus (staph. aur.)	Yellow, clot, (sterile)	Thoracic	Meningeal	Adhesions	Clinical course	
Mr. O.	40 years	Indefinite	Colorless	Lemon, no clot	Diffuse	Meningeal	Syphilitic meningitis	Clinical course	Case 3
Baby S.	2 years	1 week	Colorless	Yellow, pus	?	Meningeal	Adhesions	Clinical course	
Mr. L.	38 years	1 year	Colorless	Colorless	Thoracic	Extradural	Dislocated vertebra	Roentgen ray	
Mr. S.	32 years	2 weeks	Colorless	Colorless	Thoracic	Extradural	Tuberculosis of spine	Roentgen ray	
Mrs. M.	35 years	Few months	Colorless	Colorless	Thoracic	Extradural	Tuberculosis of spine	Roentgen ray	
Mr. P.	68 years	Months	Colorless	Colorless	?	No abnormality found at operation			

* No fluid obtained from cistern puncture, as tumor filled this area.
† Reference to previous article in Arch. Neurol. & Psychiat. 4: 529, 1920.

The pathologic findings may best be demonstrated by a consideration of four cases, chosen because of the striking manner in which the significance of the test was brought home to the examiner.

REPORT OF CASES

CASE 1.—*History.*—Mr. A., 43 years of age, had progressive paraplegia for five months. Recently there had been some numbness of the lower extremities, with an indefinite level of hypesthesia below the eighth thoracic segment and slight indication of a zone of hyperesthesia above. The gait was moderately spastic-ataxic. The blood and spinal fluid examined a short time previously had been reported "negative, with the exception of a positive globulin reaction" in the latter. In spite of the tests, syphilis was suspected, and the writer was asked to administer antisyphilitic treatment. This was not done because of the result of combined puncture the results of which are shown in Table 2.

TABLE 2.—RESULTS OF COMBINED CISTERN AND LUMBAR PUNCTURE IN CASE 1

	Cistern	Lumbar
Initial pressure	190 mm. (Same level)	150 mm.
After withdrawal of 5 c.c. from lumbar	180 mm.	60 mm.
After withdrawal of 5 c.c. from cistern	160 mm.	60 mm.
After withdrawal of 5 c.c. more from lumbar	165 mm.	0 mm.
After withdrawal of 5 c.c. more from cistern	145 mm.	0 mm.
Pulse oscillations	Just visible	Just visible
Respiratory oscillations	5 mm.	30 mm.
Rise of pressure on compression of jugular veins	300 mm.	No change
Character of fluid	Clear, colorless, no clot	Clear, colorless, no clot
Cells per cubic millimeter	0	2
Total protein per 100 c.c.	33 mg.	267 mg.
Globulin (ammonium sulphate)	0	+
Colloidal gold test	0000000000	0011100000
Wassermann reaction	Negative	Negative

Comment.—These tests indicate an unquestioned spinal subarachnoid block. If syphilis were the cause, it should in most cases be meningitic in type and the fluid should have shown cells and a positive Wassermann reaction. (Note Case 2 as a proved exception.) In a moderately progressive paraplegia with a sensory level developing, operation was clearly indicated. At operation (by Dr. W. J. Mixter) a large extradural perineurial neurofibroma was removed. The patient began to recover from the paralysis within two weeks, and is gaining rapidly at the present time.[6]

CASE 2.—*History.*—Mr. C., aged 38 years, had been treated by the writer for three years for spastic paraplegia due to unquestioned syphilis; blood and spinal fluid tests, formerly positive for syphilis, had become negative in every respect except for a slightly positive globulin reaction. For six months past he had become more spastic and complained now of numbness to the waist. The findings were marked paraplegia with sensory diminution below the tenth thoracic segment. The results of combined puncture are shown in Table 3.

Comment.—These hydrodynamic tests are not so convincing in description as on observation. A slight obstruction in the lumbar needle could cause the changes found, especially the slow rise and fall in the lumbar needle following compression of the jugular veins, but this test was repeated both before and after withdrawal of fluid with the same result each time. It seemed that with

6. Combined puncture has recently been repeated and now shows no block.

the change in symptomatology there might also be a change in pathology. Operation was therefore performed by Dr. Mixter and a well-marked diffuse swelling of the spinal cord was found, which on microscopic section of an excised piece showed typical gumma. It is probable that the formation of gumma was coincident with the increase in symptoms of cord compression, and it is important to note that it developed in a patient in whom Wassermann tests had become negative; also that neither before nor after the operation did the latter symptoms yield to antisyphilitic treatment.

Case 3.—*History.*—Mr. O., 40 years of age, presented symptoms suggestive of root pain in the right thorax, intermittent for the past five years. There were no constant symptoms or signs of cord compression. A xanthochromic lumbar fluid giving a positive Wassermann reaction had been obtained. In

TABLE 3.—Results of Combined Puncture in Case 2

	Cistern	Lumbar
Initial pressure	90 mm. (Same level)	80 mm.
After withdrawal of 10 c.c. from lumbar	90 mm.	50 mm.
After withdrawal of 5 c.c. from cistern	60 mm.	50 mm.
Oscillations in manometer, pulse	Good	Almost none
Oscillations in manometer, respiration	Good	Slight
Increase of pressure on jugular compression	100 cm. prompt rise	100 cm. slow rise
Fall of pressure on jugular compression	Prompt fall	Slow fall
Character of fluid	Clear, colorless, no clot	Clear, colorless, no clot
Cells per cubic millimeter	28	8
Total protein per 100 c.c.	68 mg.	178 mg.
Globulin	+	++
Wassermann reaction	Negative	Negative
(Blood Wassermann reaction negative)		

TABLE 4.—Results of Combined Puncture in Case 3.

	Cistern	Thoracic 12	Lumbar 4
Character	Clear, colorless no clot	Light yellow, no clot	Darker yellow, no clot
Cells	6	24	—
Total protein per 100 c.c.	42 mg.	869 mg.	952 mg.
Globulin	0	++	++
Colloidal gold test	0000000000	0000012100	0000012200
Wassermann test	+++	+++	+++

this case it was thought possible that a syphilitic meningeal block was present, and therefore it was decided to determine whether this could be demonstrated by differential puncture. The findings from puncture of three loci of the subarachnoid space are given, the needles being introduced into a cistern, into the twelfth thoracic and fourth lumbar spaces, respectively. The readings following withdrawal of fluid are not satisfactorily recorded but indicated block. The effect of jugular compression was most instructive; a prompt rise was shown in the cistern manometer; after the pressure had risen about 100 cm. the fluid began to rise in the thoracic manometer, and later still in the lumbar. The fall in pressure on release of the jugular veins was in the same order, prompt in the cistern, slow in the thoracic and slower still in the lumbar manometers. A striking difference is also to be seen in the examination of the three fluids.

Comment.—The examination apparently demonstrated by physical and chemical procedures that the subarachnoid space is here separated into compartments, not freely intercommunicating, and appears to corroborate the

diagnosis of a diffuse syphilitic meningitis. Under antisyphilitic treatment the patient rapidly improved and two months later combined puncture (cistern and fourth lumbar space) gave a very different picture.

Comment.—This examination in no way resembles that before treatment. It is reasonable to think that under treatment the obstruction to the free passage of fluid in the spinal meninges has been removed.

Another combined puncture has recently been repeated in the case of this patient, the findings being essentially as last recorded, namely, no block was demonstrated. During the interval—about six months—the patient has been clinically well.

TABLE 5.—RESULTS OF PUNCTURE IN CASE 3 AFTER ANTISYPHILITIC TREATMENT

	Cistern	Lumbar 4
Initial pressure	185 mm. (Same level)	135 mm.
After withdrawal of 5 c.c. from lumbar	140 mm.	100 mm.
After withdrawal of 5 c.c. from cistern	115 mm.	75 mm.
Oscillations with pulse	Good	Fair
Increase on jugular compression	Prompt rise and fall	Prompt rise and fall
Character of fluid	Clear, colorless	Clear, colorless
Cells	4	8
Total protein	27 mg.	67 mg.
Globulin	0	0
Sugar	0.046 per cent.	0.046 per cent.
Wassermann test	.....	Weakly positive

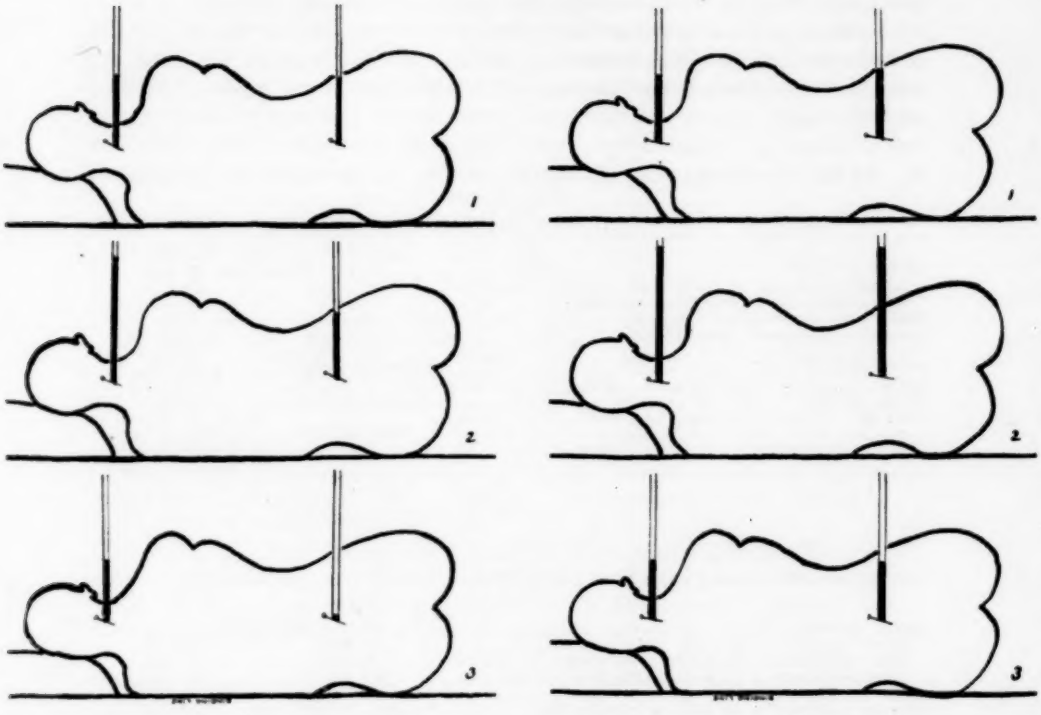
TABLE 6.—RESULTS OF COMBINED PUNCTURE IN CASE 4

Sept. 21, 1920	Cistern	Lumbar
Initial pressure	130 mm. (Same level)	140 mm.
After withdrawal of 6 c.c. from lumbar	130 mm.	0 mm.
After withdrawal of 10 c.c. from cistern	50 mm.	0 mm.
After injection of 9 c.c. of saline into lumbar sac	100 mm.	180 mm.
Pulse oscillations	Good	Slight
Change of pressure on jugular compression	Prompt rise to 200 mm.	No rise
Character of fluid	Clear, colorless no clot	Clear, colorless, no clot
Cells	3	1
Protein (alcohol precipitation)	Normal	Marked increase

The following case is perhaps the most instructive, because operation for spinal cord tumor would not have been undertaken at the early date on which it was performed but for the demonstration of subarachnoid block.

CASE 4.—*History.*—R., 13 years of age, presented an apparently typical right hemiparesis, involving the face, arm and leg, but he had also a slight deltoid atrophy. There was no objective sensory disturbance at this time or subsequently. Shortly afterward spasticity appeared in the left leg also, and for this reason the possibility of cord tumor was considered and combined puncture performed.

The examination indicated subarachnoid block, but as the method was at that time new, and there was so little guide as to the location of the block, the patient was observed for two weeks, during which time slight spasticity began in the left arm. At this time (October 8) combined puncture was repeated, with the same findings as before. On this second examination total protein quan-



A B

A, diagrammatic representation of some of the findings in Case 4, showing spinal subarachnoid block. 1, initial pressures, on same level; 2, effect of jugular compression. Pressure rises promptly in cistern, not at all in lumbar manometer. 3, effect of withdrawal of 6 c.c. of spinal fluid from lumbar sac. Pressure here falls to zero; pressure in cisterna magna unaffected. The lumbar fluid contains an excess of protein; that from the cistern is normal.

B, same case as in A. Tests repeated two months after removal of extradural tumor. 1, initial pressures; 2, effect of jugular compression; 3, result of withdrawal of 5 c.c. of fluid from lumbar sac. The fluids in both manometers now maintain the same level under all conditions. The two fluids are now similar in character.

titation showed 21 mg. per 100 c.c. in cistern fluid, 85 mg. in the lumbar. Operation was now undertaken (Dr. C. A. Porter), our chief guide as to level being the deltoid atrophy. An enchondroma, originating from the intervertebral disk, presumably of the fifth cervical vertebra, was found deeply indenting the dura mater and the underlying cord.

Following the removal of this tumor the patient rapidly regained his power. On December 15 another combined puncture was performed, the results of which are shown in Table 7.

Comment.—The postoperative findings coincide in every detail with our conception of normal relations of cistern and lumbar fluids. The examinations in this case are invaluable in determining what is normal and what is pathologic, and consequently what criteria are to be relied on in our estimation of subarachnoid block. In this case, moreover, it is not too much to say that we had at hand a diagnosis of block before a diagnosis could be made from clinical examination and that when operation was performed one of the most important clinical signs—a level of sensory change—was still entirely absent. It is well also to emphasize the unusually rapid improvement following operation, due as much to the early diagnosis as to the operative skill.

TABLE 7.—RESULTS OF POSTOPERATIVE COMBINED PUNCTURE IN CASE 4

	Cistern	Lumbar
Initial pressure	190 mm. (Same level)	160 mm.
After withdrawal of 5 c.c. from lumbar	160 mm.	130 mm.
After withdrawal of 5 c.c. from cistern	130 mm.	95 mm.
Oscillation with pulse	Good	Fair
Change of pressure on jugular compression	Prompt rise and fall	Prompt rise and fall
Character of fluid	Clear, colorless no clot	Clear, colorless, no clot
Cells	0	0
Total protein per 100 c.c.	29 mg.	33 mg.
Globulin	0	0

The findings in this case are as convincing as a well-conducted physiologic experiment. We find a fluid which definitely demonstrates a block. At operation the dura and its contents are found "knuckled" and displaced; this is relieved without opening the subarachnoid space, and two months later we find a free passage and entirely normal fluids above and below.

EVIDENCE OF STASIS ALONE

Two patients showed no evidence of obstruction to the free passage of spinal fluid in the subarachnoid space, and yet marked difference in protein was found in cistern and lumbar fluids; in fact, the difference was more marked than in some cases in which an hydrodynamic block was observed. One of these patients presented a vertebral myeloma at operation (cistern 100 mg., lumbar 250 mg. of protein); the other subdural neurofibroma at necropsy (antemortem fluids, cistern 30 mg., lumbar 135 mg.). The question arises, How much reliance shall be placed on the findings of excess protein alone in the lumbar sac? Can we say that excess in the lumbar fluid twice that of the cistern, or

thrice or four times indicates cord compression? We are obviously on much less certain ground in our reasoning in the absence of a block dynamically shown to be present.

In order to make any statement on this point it will be necessary to know to what extent conditions other than cord compression can alter the normal chemical findings in the fluids drawn from above and below, and this will require more data than are at present available. In this series of seventy-one punctures the lumbar protein estimation has been found to be twice that of the cistern fluid in several cases of multiple sclerosis and in one case of undoubted acute poliomyelitis. In no case has a difference of more than this been noted in patients presenting an uncomplicated degenerative or exudative myelitis. This whole subject of variation in the character of the spinal fluid associated with nonobstructive lesions is intensely interesting and important, but one which will require much further study.

CASES PRESENTING NO EVIDENCE OF HYDRODYNAMIC BLOCK OR STASIS

We have already considered this group and come to the conclusion that absence of block as demonstrated by the foregoing procedures is the normal state. The important question now arises, Can we have cord compression with fluid findings normal in every respect? Certainly we can. This was proved in one case of this series. The patient was a man who had been paraplegic for ten years, with a well-defined sensory level of anesthesia. The tests were normal in every way, the two fluids being identical on examination. At operation an elongated epidural multilocular cyst was found. In a considerable number of cases examined as to lumbar fluid alone normal protein values have been obtained in a few; as protein excess seems to antedate demonstrable block, it is likely that combined puncture would in these patients have showed normal dynamic relations. It is interesting to note that operation in these cases has in the experience of the writer never been of value in restoration of function.

SUMMARY AND CONCLUSIONS

For some years valuable data as to the presence of spinal cord compression have been obtained from examination of the lumbar fluid. The actual value of these tests was, however, often only confirmatory, and at times purely academic, for by the time a fluid could be called pathognomonic or even strongly suggestive of cord tumor myelitic changes were usually so marked as to negative the effect of an otherwise successful operation.

By means of double punctures certain writers of the French school have obtained information of value, but their method of approach, that

is, puncture of thoracic spine, etc.,[7] is hardly applicable to systematic study of doubtful cases; nor do these writers appear to have laid sufficient stress on the importance of pressure changes under different conditions, relying chiefly on the less important laboratory examination of the fluids obtained.

By the use of combined cistern and lumbar puncture it is possible not only to obtain fluid above and below a supposed cord lesion, but also to analyze the mechanical factors involved in the flow of the fluid, and thereby estimate the permeability of the subarachnoid space. Where obstruction has been demonstrated by this means, an adequate explanation has been found in seventeen of eighteen cases. The importance of the procedure lies in the fact that definite evidence of pathology is forced on the investigator early in the process of compression, and that if advantage is promptly taken of the findings a relatively intact cord may be saved. Hence this method may show obstruction in patients in whom compression seems unlikely, whereas the chemical findings from lumbar puncture alone are usually not sufficiently striking until a diagnosis of cord compression is clinically apparent.

It is not to be thought that every cord tumor will present the picture of subarachnoid block, but it is clearly shown that cord tumor frequently presents a block very early. Hence the positive findings are of great value. Negative findings are of relative importance.

In that cistern puncture requires considerable practice and is a potentially dangerous procedure, it is fortunate that certain hydrodynamic studies of value may be carried out on the lumbar fluid alone. Most significant of these is the observation of the pulse oscillations and change of pressure on artificially increasing cerebral pressure by compression of the jugular veins. These observations call for a return to the use of the original aqueous manometer, which in the writer's opinion should be employed in every diagnostic lumbar puncture.

NOTE.—Not until this paper was finished was my attention called to a paper by Queckenstedt[8] in which he writes of the significance of jugular compression in spinal cord tumor. His conclusions are amply confirmed by my observations. It is, however, obvious that by the use

7. Marie, Foix and Bouttier: Double punction sur-et-sous-lèsionelle dans un cas de compression medullaire: xanthochromie, coagulation massive dans le liquide inferieure seulement, Rev. Neurol., 1914, p. 315; Ravaut and Krolunitsky: Oreillons et meningite cerebro-spinale a paramenigo-cocques. Guerison par injections intrarachidiennes lombaires et cervicales, Soc. méd. des hôp. de Paris **39**:618, 1915. Souques and Lantuéjoul: Hyperalbuminose enorme du liquide cephalo-rachidien dans un cas de coagulation massive, Rev. Neurol., 1920, p. 137. Marie, Foix and Robert: Service que peut rendre la punction rachidienne pratiquée a des etages differents pour le diagnostic de la hauteur d'une compression medullaire, Rev. Neurol., 1913, p. 712.

8. Queckenstedt: Ztschr. f. Nervenheilk, **55**:325, 1916.

of the combined puncture slight changes in dynamic tests and slight differences in chemical composition of the two fluids must be of greater significance than abnormalities in the lumbar fluid alone.

DISCUSSION

Dr. Henry Viets of Boston stated that before the war he had the pleasure of working with Dr. Ayer in some of his early work on spinal fluid, although he played a very minor rôle in that work. Since then he had watched with the greatest interest Dr. Ayer's subsequent studies. He believed that Dr. Ayer, in his work on cistern puncture and subarachnoid block, had made a most valuable contribution to neurology, especially helpful in the diagnosis of spinal cord tumors and in the treatment of syphilis of the nervous system, and meningitis.

Dr. Viets stated that he had found some doubt in the minds of men throughout the country regarding the advisability of cistern puncture because of its well-known dangers. He felt, however, that in experienced hands, there was little danger in this operation and that one was always surprised, after one or two punctures had been made, at the ease with which this procedure could be carried out.

The value of cistern puncture was emphasized in a recent case of meningitis in which numerous spinal punctures had failed to obtain fluid and there was an obvious condition of subarachnoid block from meningitis. As the patient rapidly became worse, a cistern puncture was performed with the release of a large amount of purulent fluid, and antimeningitis treatment was given in the cistern at the same time. The man made a rapid recovery after this procedure.

Dr. Charles A. Elsberg of New York said that he had had no experience with cistern puncture; in fact, he stated that he had been rather afraid of it. If left in the hands of one as expert as Dr. Ayer to work out the method, it might be developed into a relatively safe procedure, and then it might be useful in certain classes of cases.

In answer to a question by Dr. Schwab regarding the possible surgical objections to this method, Dr. Elsberg said that Dr. Ayer could answer the question better than he. Dr. Elsberg's objection, which was theoretical and perhaps entirely wrong, was that the degree of distention of the cisterna varied according to the patient and condition. In suboccipital operations he had often seen the cisterna magna distended with fluid so that it probably could be punctured with safety, but in many instances, the cisterna was small—not more than 2 cm. wide and from 0.25 to 0.5 cm. in depth. In such a small cistern, he had wondered how great the danger of cistern puncture would be.

Dr. Ernest Sachs of St. Louis said he had been very much impressed with the careful work that Dr. Ayer had done in this direction and unquestionably the procedure seemed very safe in his hands. Dr. Sachs said he had always been impressed with the fact that whenever he exposed the medulla there was marked movement of the medulla with each respiration. It was his belief that the basal cisterna was there so that the medulla would have ample room to move back and forth with each respiration. It seemed to him that this might be a possible source of danger.

Dr. Sachs asked whether Dr. Ayer's procedure would positively prove in which cases there was a block to the cerebral spinal circulation; if this were possible, the cases of so-called myelitis which had no block would not need exploratory operations, and this would be of inestimable value to patients.

In last year's meeting of the American Neurological Association he brought up a point which seemed to Dr. Sachs a real danger. He pointed out that the circular vein which runs around the foramen magnum might be punctured as it sometimes extended below the foramen magnum. Dr. Cushing attacked him rather violently and said this vein never extended below the foramen magnum.

Dr. Sachs had one case in the past year of a child with a cerebellar tumor, in which this circular vein was enormously enlarged and extended up about a centimeter above the foramen magnum and also below, and he was quite sure that in this particular case had a puncture been done, the sinus (it was a sinus; it was no longer a vein) would unquestionably have been hit with the needle.

Dr. Simon P. Kramer of Cincinnati said that Dr. Ayer's work on puncture in the cistern had again proved that lumbar anesthesia should be abandoned, and that there should be no intraspinal medication with anything in the nature of a neural poison.

So far as the danger of cistern puncture was concerned, he agreed with Dr. Ayer, that there was no more danger from subarachnoid punctures than from those in the lumbar region. The same danger, of course, existed there. Dr. Ayer had spoken of the fact that puncture should not be performed when one suspected an intracranial tumor. In all other cases the procedure was just as safe as a puncture of the subarachnoid space in any other part of its course.

Dr. Charles Elsberg of New York, apropos of Dr. Kramer's remarks, asked whether Dr. Kramer meant to say that a puncture in the lumbar region below the lower end of the cord and between nerve roots was no less dangerous than a puncture anywhere along the spinal canal where there was cord, and especially high up where the medulla was situated. He asked whether Dr. Kramer meant that there was no more danger when the medulla lay in front of the needle than when there were only nerve roots.

Dr. Henry A. Cotton of Trenton, N. J., stated that during the past year over 200 cisterna punctures had been performed in the State Hospital at Trenton without any ill effects. They had substituted it temporarily for the intracranial treatment in paresis, and so far as he had been able to see there was less reaction from a cistern puncture than from a lumbar puncture; the patients really preferred it. Those who were intelligent asked for a "neck puncture" as they called it, rather than a lumbar puncture. It is a valuable method, and in time it will be a great improvement over the lumbar method of treatment. The method can be used with safety, but like all technic must be done correctly. There was no danger whatever, as far as he was able to judge, from the 200 punctures which had been performed.

Dr. James B. Ayer of Boston, in closing, said that he realized that the question of safety in carrying out cistern puncture was of paramount importance. Unquestionably the procedure was potentially dangerous, but if a person familiar with lumbar puncture proceeded cautiously, with due attention to detail of needle, position of patient, etc., he believed it safe. Even if the median vein of which Dr. Sachs spoke were present, he questioned whether a blunt needle would perforate it. As a matter of fact about 500 cistern punctures had been performed by a number of workers so far without serious trouble, and it is now being employed at the Massachusetts General Hospital more and more frequently, not as a substitute for lumbar puncture, but where indicated, usually in spinal subarachnoid block.

Concerning the movement of the medulla spoken of by Dr. Sachs, he said that before the method had been used on the living, several cadavers had been examined. Needles were placed in the cisterna and brain removed above

the pons; in these subjects it was possible to flex, extend and rotate the head without causing the medulla to jam backward and impinge the needle. It would be expected that in some cases of brain tumor the relations might be such that the medulla might obliterate the cisterna magna; these cases should be studiously avoided.

As for the suggestion that because cistern-lumbar puncture does not render positive findings in every case of cord tumor it is therefore worthless, it seemed to him no argument at all. That it usually gave information of value very early should outweigh the fact that it sometimes failed.

In concluding, Dr. Ayer emphasized his change of attitude toward cord tumor cases in view of the findings of subarachnoid block. Previously, in his experience, operation was seldom undertaken unless a diagnosis was almost certain. Now the presence of subarachnoid block was forced on the examiner, at times long before a diagnosis of cord tumor could be more than just entertained as a possibility. By using this method the necessity for operation has been forced on the mind much earlier, and in a number of patients the prognosis has accordingly been much brighter.

ACUTE BENIGN MENINGO-ENCEPHALITIS WITH PAPILLEDEMA

FOSTER KENNEDY, M.D.
NEW YORK

The following cases are grouped because they have many factors in common, together making a picture which, as far as I can discover, has not been made plain in the literature on inflammations of either the meninges or brain substance. None of them has the general coloring which we have learned to associate with epidemic encephalitis, but the fact that they have come to notice during the period of incidence of that disease and were not familiar to us before that time is enough to make one cautious about asserting that the two conditions are entirely unrelated. My patients all had evidence of systemic infection, as shown by the presence of a changed blood picture, fever and general malaise. The onset was acute—in some cases sudden, with headache serving as an inadequate warning of trouble to come. In all, a period of stupor was followed by one of excitement or disorientation, which lasted only a few days in most instances, to be followed, as a separate episode, by focal cerebral palsy-hemiplegia, hemianopia, aphasia or cranial nerve inadequacy. The rushing onset of optic neuritis late in the illness, synchronizing with amelioration of symptoms previously acquired, and its rapid amelioration in turn, are phenomena with which I, at least, have been unacquainted, and must surely depend, for their production, on sudden blockings of intraventricular drainage by meningitic exudate, and for their disappearance, on a reconstitution of a normal fluid mechanism.

REPORT OF CASES

CASE 1.—*History.*—L. C. P., aged 35, admitted to Bellevue Hospital on Feb. 2, 1921, had had headache for a week previously and a frequent twitching of the left side of the face. On Feb. 2, he left his partner when apparently otherwise in normal health, went into a subway train, became noisy and was therefore ejected from the train; became restless, yawned often and passed into unconsciousness. His tongue was coated, dry and lacerated, and the uvula was covered with a white membrane. His pharynx was congested and his neck was rigid. Kernig's sign was present. The pulse rate was 120, and his temperature was 104. The systolic blood pressure was 150. Spinal fluid: raised pressure, clear, 70 cells (98 per cent. lymphocytes). Globulin was increased, and the Wassermann test was negative. The colloidal gold test showed normal precipitance. The Wassermann test of the blood was negative; a blood culture was negative. There were 17,000 white blood cells per cubic millimeter of which 82 per cent. were polymorphonuclears. There was incontinence of urine and feces. He perspired profusely. There was punctate erythema over the back and buttocks which faded in a few days. The throat culture was that of *Staphylococcus*

aureus. Blood chemistry examination revealed a slight nitrogen retention and, oddly enough, a low blood sugar content. The fundi were normal. The abdominal reflexes were absent. There was a Babinski sign on the left.

Course.—Two Days Later: The temperature was 100. He was no longer unconscious, but was entirely amnesic for the events of his illness. He was rational except for short periods at night. He had a severe headache and was drowsy, but he was no longer incontinent. On the preceding day he became conscious and was violent, requiring restraint.

Three Days Later: The rash was disappearing. His temperature was normal in the morning; 100 at night. He was drowsy, restless and had twitching of the limbs and fingers. He complained of sensitiveness over the left auricle and left side of the face. Examination of the ears was negative. He yawned frequently.

Two Days Later: His speech was not clear. There was evidence of anomia. He was fluent but incorrect in naming seen objects, and was embarrassed by his mistakes. He continued in perseveration of his errors to his own confusion.

Eighth Day of Illness: He was stuporous aud refused to talk. His head was retracted. Kernig's sign was more positive. Examination of the ear was negative. There was tenderness over the left temporoparietal area and auricle. The spinal fluid contained 81 cells (lymphocytes 92 per cent.); culture and guinea-pig inoculation were negative; no tubercle bacilli were found.

Ninth Day of Illness: The stupor had largely disappeared, and he was rational. He was well oriented but presented a complete picture of temporosphenoidal aphasia, together with a central weakness of the right side of the face. A radiogram of the skull at this date was negative for sinus disease or fracture.

Tenth Day: In the morning he had fluent anomia, weakness of the entire right side and hemianesthesia including the right cornea. There was ankle clonus on the right. There were 20,000 white blood cells of which 81 per cent. were polymorphonuclears. He was restless and tried to get out of bed. His temperature was never higher than 100 F. at night and was usually normal by day. The fundi were normal.

Twelfth Day: His condition was practically unchanged. The relative weakness of the right arm and leg had decreased, but drowsiness was great and the aphasia unchanged. The fundi were still normal.

Fourteenth Day: Hemianesthesia was less evident. Drowsiness was severe. Occasionally he named objects correctly. On the next day no sign of sensory defect could be made out. The aphasia was serious but it was improving. Drowsiness was less severe. The fundi were normal; owing to the obvious suspicion that he had a brain abscess, these were examined daily, despite daily improvement in the pyramidal abnormality and aphasia.

Eighteenth Day: On this day, when all sensory defect was gone, the right hemiplegia reduced to a weakness of the lower part of the right side of the face and absence of the right abdominal reflexes, and when the aphasia though still severe, was clearly improving, I was astonished to find the nerve heads distinctly hyperemic, the margins blurred and linear striate hemorrhages on the retinae. Within twenty-four hours there was a swelling on each nerve-head of 4 diopters which shortly afterward became 6; the veins became engorged and tortuous, and splashing hemorrhages were seen around the disk and on the papilla itself. From that time on there was a constant improvement in the focal signs and symptoms, but the fundal state did not change for four and a half weeks; then rapid improvement and absorption of hemorrhages began so that restitution of

normal conditions in the eye as regards appearance and function was complete in ten days. The patient is now perfectly well, with the exception of some naming difficulty when fatigued.

CASE 2.—*History.*—E. O., a pregnant woman aged 30, had lobar pneumonia on Jan. 23, 1921. Five days later labor began. Presentation was impossible and embryotomy was performed. On the eleventh day of the pneumonia the fever had disappeared, and the patient seemed to be having normal convalescence. On February 6, she complained of severe headache and tenderness over both mastoids; the temperature rose to 100 F., and the pulse rate fell to 50 from 80. The next morning she was stuporous and had a left hemiplegia; she was restless, almost violent, and noisy. Next morning she was in coma, the temperature was 103 F., the pulse rate 100, and slight blurring of the disks was thought indefinitely to be present on both sides, but especially on the left. On that day she had a dozen focal convulsions, beginning on the left side of the face, and at this end involving both sides equally. On the second day of hemiplegia the blood count was 12,000 white cells, of which 77 per cent. were polymorphonuclears. On the third day there were 23,800 white cells, of which 83 per cent. were polymorphonuclears.

The spinal fluid contained only 7 cells and was normal in all its reactions. Five days after its onset, the hemiplegia began to clear up, the leg recovering first. Consciousness returned slowly, and the patient could repeat a few words after her husband. The temperature was 101 F., however, and a few hours after regaining consciousness she developed visual hallucinations, and was violent and disoriented for five days, after which she was mentally clear and emotionally placid. During this stage the spinal fluid was normal, except that it was under pressure and had a mononuclear content of 12.

Course.—Twelve days after the onset of her hemiplegia, the temperature, pulse and respirations were normal. She had headache, especially over the right posterior parietal region, but the palsy had disappeared from the face and leg and was improving daily in the left arm. The optic disks were moderately blurred, the margins, however, being clearly seen, and no swelling capable of measurement was present. On the morning after this fundal observation, she complained of increased headache and of not being able to see properly. The right retina was then found to be the seat of several large hemorrhages, and on the left were smaller hemorrhages; these increased in size and number in both eyes for seven days. Edema was 6 diopters in height, and no disk margin was visible on either side. In this attack of optic neuritis there was no added symptom beyond increased headache. Vision on the third day was reduced to 20/60 and paracentral scotomas were present. On the seventh day after the appearance of retinal hemorrhage, the swelling of the papillae began to subside, the scotomas to decrease, the retinal blotches to contract and vision to return to normal. At the present time this patient is without symptoms, but the disk edges are not quite clear, though papilledema is negligible and vision normal.

CASE 3.—*History.*—Mrs. R., aged 67, was seen by me in consultation with Dr. Lloyd of Brooklyn in November, 1919. Her illness began two weeks before my visit, with headache, moderate fever and intense pain in the back of the neck. After some days she had moderate ptosis of the left eyelid which rapidly became complete, and to which was added palsy of all ocular muscles served by the left third nerve. She had no lethargy—there were no symptoms referable to a periaqueductal lesion. Fever never rose higher than 101 F., and the temperature was normal after the third week. There was slight rigidity of the neck and a moderate Kernig's sign. The spinal fluid, in the third week of

illness, contained 70 lymphocytes and was bacteriologically negative, and negative also to the Wassermann test. In this same week she began to complain of numbness and tingling in the right hand and to have a distinct naming difficulty, which in a week's time developed into an absolute temporosphenoidal aphasia of fluent type. She then became violently excited, screamed often and long, was disoriented and harbored constant vague persecutory delusions. The fundi were normal throughout her illness. The condition remained practically unchanged for three months, when rapid improvement occurred, so that in three weeks or less she made only occasional errors in speech, and there was only a slight ptosis of the left eyelid. This patient is now well, though upward movement of the left eye has not returned, and her speech is normal. No outspoken right pyramidal signs ever developed. The position of the lesion, which I presumed to be a localized meningitic exudate, must have been basal and must have included the left third nerve as it winds around the crus, and also the adjacent temporosphenoidal lobe. She complained throughout her illness of pain and tenderness in the left temple. A careful radiographic examination showed the sphenoidal and other sinuses to be free from disease. The localized bony tenderness here is to be compared to the left auricular and facial tenderness of the first case and the right posterior parietal tenderness of the second. Kidney function tests were normal as was the blood chemistry for nitrogen and sugar. A leukocytosis of 15,000, of which 65 per cent. were polymorphonuclears, obtained in the second week of her illness.

Case 4.—*History.*—A. B., a woman aged 46, complained in December, 1920, of insomnia, headache and a feeling of mental confusion. In January she became disoriented and noisy, and was admitted to the psychopathic department of Bellevue Hospital. Her blood pressure was: systolic 290, diastolic 70, but no renal defect could be discovered, though the fundal eye vessels were distinctly sclerotic. After a month her mental condition was again normal, but on March 3, she was stuporous with paresis of the left side, had half-sided pathologic reflexes and was incontinent of urine with some impairment of speech and orientation. There was acute tenderness over the right parietal area. The spinal fluid contained 29 cells per cubic millimeter and was culturally negative. The Wassermann test was negative in the blood and spinal fluid.

Course.—Three days later the fundi presented the appearance described in the other cases—profuse hemorrhages splashed on and around the disk which was itself acutely swollen. She was garrulous and unaware of her surroundings; she vomited and was incontinent. These symptoms lasted until March 21, and to them was added a complete left hemianopia. Amelioration of all abnormal conditions then began; inside a week she was clear mentally, felt and looked well, and the left hemiparesis and hemianopia had quite disappeared. The papilledema had almost disappeared, and the retinal hemorrhages were undergoing absorption. In May, 1921, she presented no signs or symptoms of her illness, though essential hyperpiesia was maintained as before her acute condition occurred.

Case 5.—*History.*—C. K., aged 20, in February, 1920, had severe left-sided headache for two days, after which she awoke in the night and found herself unable to move the right arm or leg. She became frightened, screamed loudly, became unconscious and remained so for two days and nights. On recovering consciousness she was excited, required restraint and was completely aphasic. These conditions, together with urinary incontinence, lasted for twelve days, after which she became rational again and the aphasia rapidly improved. She was admitted to the neurological department of Bellevue Hospital on March

Case 4.—These fundal phenomena developed inside twenty-four hours during the tenth week of illness. They persisted for only seventeen days and then began rapidly to improve so that the fundi again became normal. (Drawn by Dr. Fairbanks, Bellevue Hospital.)

Illustrating Article by Foster Kennedy, M.D.
Archives of Neurology and Psychiatry, January, 1922

8, 1920, for "nervousness and right hemiplegia." She had no trace of aphasia then; the hemiplegia was only relative in degree and most evident in the right leg. The fundi—normal on admission—were covered two days later with hemorrhages, and the papillae were invisible through swelling. On this day she had severe left parietal headache; she vomited and was restless and uneasy. Lumbar puncture showed a fluid under increased pressure, of a light yellow color, thicker than usual and containing 20 lymphocytes. Papilledema was at its height in three days from its inception; it then rapidly retrogressed and in ten days no swelling could be made out. In three weeks all trace of the hemorrhages had also disappeared. This patient today—fifteen months after her illness—is normal except for a spastic cerebral paresis of the right lower extremity.

CONCLUSION

One cannot view the rapidly fluctuating syndromes of these cases, of true epidemic encephalitis, or of acute disseminated sclerosis, without being impressed by the rôle played by edema in cerebral lesions, and the different prognosis produced by mere physiologic block on the one hand and by obliteration of structure on the other.

A MORE ACCURATE CLINICAL METHOD OF DIAGNOSIS OF PERIPHERAL NERVE LESIONS AND OF DETERMINING THE EARLY RECOVERY OF A DEGENERATED NERVE

WITH REPORT OF CASES AND EXPERIMENTAL DATA *

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Introduction.
Description of instrument by Professor Pyle.
Physiologic principles of muscle and nerve muscle reactions based on a consideration of the literature.
Clinical application of chronaxie.
Discussion of other methods.
Relation of induced waves of opening and closing.
Experimental data.
Clinical cases.
Discussion.
Summary and conclusions.

INTRODUCTION

Since surgeons and neurologists primarily have had to deal with the large number of peripheral nerve injuries resulting from the late war, there has been an unusual number of investigations carried on to determine the underlying principles of nerve regeneration and the best methods of treating them. The physiologic and anatomic features of nerve degeneration, as well as the surgical methods to correct them, have been studied particularly by Howell,[1] Huber,[2]

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* This paper was read in abstract before the American Neurological Society, June 14, 1921, Atlantic City.

1. Howell and Huber: A Physiological, Histological and Clinical Study of the Degeneration and Regeneration in Peripheral Nerve Fibers After Severance of Their Connection with the Nerve Centers, J. Physiol. **13**:335, 1892; **14**:1, 1893.

2. Huber, G. C.: A Study of the Operative Treatment for Loss of Nerve Substance in Peripheral Nerves, J. Morphol. **11**:630-733, 1895; Transplantation of Peripheral Nerves, Tr. Chicago Path. Soc. **11** (June 1) 1919; Repair of Peripheral Nerve Injuries, Surg., Gynec., & Obst. **30**:464 (May) 1920.

Ranson,[3] Vanlair,[4] Kilvington,[5] Sherren,[6] Lewis,[7] Langley,[8] Pollock,[9] Tinel,[10] Athanassio Bénisty [11] and others. Few of the researches have combined the physiologic and anatomic methods. The problem, however, on which little advance has been made is the determination of whether a paralyzed peripheral nerve has any regenerative power. To the surgeon this question is of vital importance, for, though we now know that secondary nerve sutures when carried out by the newer methods afford a much better prognosis than was formerly supposed, a nerve suture should be undertaken at the earliest possible moment. Therefore, if it were possible to determine earlier than heretofore that a paralyzed nerve is beginning to regenerate, a great advantage would be gained.

Keith Lucas [12] and Lapicque [13] demonstrated on cold blood animals that there were certain striking differences between muscles with intact

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3. Ranson, S. W.: Degeneration and Regeneration of Nerve Fibers, J. Comp. Neurol. **22**:487, 1912; Non-Medullated Nerve Figers in Spinal Nerves, Am. J. Anat. **12**:67, 1911.

4. Vanlair, C.: De la néurotization du cartilage osseux dans la suture tubulaire des nerfs, Arch. de physiol., norm. et path. **9-10**:595, 1882.

5. Kilvington, B.: An Investigation on the Regeneration of Nerves, Brit. M. J. **1**:935 (April) 1905. Osborne, W. A., and Kilvington, B.: Axon Bifurcation in Regenerated Nerves, J. Physiol. **37**:1, 1908. Kilvington, B.: An Investigation on the Regeneration of Nerves with Regard to Surgical Treatment of Certain Paralyses, Brit. M. J. **1**:1414, 1908.

6. Sherren, J.: Injuries of Nerves and Their Treatment, New York, William Wood & Co., 1908.

7. Kirk, E. G., and Lewis, D. D.: Regeneration in Peripheral Nerves, Bull. J. Hopkins Hosp. **28**:71 (Feb.) 1917; Fascial Tubulization in Repair of Nerve Defects, J. A. M. A. **65**:486-491 (Aug. 7) 1915.

8. Langley, J. N., and Anderson, H. K.; On Autogenetic Regeneration in the Nerves of the Limbs, J. Physiol. **31**:418, 1904. Langley, J. N.: Observations on Denervated Muscle, J. Physiol. **50**:335, 1915-1916; On the Separate Suture of Nerves in Nerve Trunks, Brit. M. J. **1**:45 (Jan.) 1918.

9. Pollock, L. J.: The Clinical Signs of Nerve Injury and Regeneration, Surg., Gynec. & Obst. **30**:472 (May) 1920.

10. Tinel, J.: Nerve Wounds (Monograph), New York, William Wood & Co., 1917.

11. Bénisty, Mme. Athanassio: Formes cliniques des lesions des nerfs, Monograph, Paris.

12. Lucas, K.: On the Optimal Electric Stimuli of Normal and Curarized Muscles, J. Physiol. **34**:372, 1906; On the Optimal Electric Stimuli of Muscle and Nerve, ibid. **35**:103, 1907; The Analysis of Complex Excitable Tissues by Their Response to Electric Currents of Short Duration, ibid. **35**:310, 1907; On the Rate of Development of the Excitatory Process in Muscle and Nerve, ibid. **37**:459, 1908.

13. Lapicque, L.: Sur la theorie de l'excitation electrique, J. de physiol. et de path. gen. **10**:601, 1908; Conditions physiques de l'excitation electrique, J. de physiol. et de path. gen. **11**:1009, 1909; Techniques nouvelles pour l'electrodiagnostic, Compt. rend. Acad. d. sc. **161**:643 (Nov. 22) 1915; Presentation d'un chronaximétre clinique, Compt. rend. Soc. de biol. **78**:695, 1915.

nerve supply and those without any. Adrian [14] confirmed this on human muscles and showed, as we shall see later, that a muscle whose nerve has degenerated requires a current of longer duration to cause a contraction than does a muscle with normal nerve supply, and that all currents are of far shorter duration than can be obtained with ordinary apparatus. To obtain these very short currents Lucas [12] devised his pendulum and Lapicque [13] his "chronaximeter."

For these reasons, therefore, we undertook some experiments on sutured nerves to determine whether by applying the principles laid down by Lucas, Adrian and Lapicque for differentiating normal and degenerated nerves we might throw some light on these questions.

It at once became apparent, however, that there were certain inherent objections to the Lucas pendulum for clinical purposes. It is very heavy and practically nonportable, expensive in construction and difficult to keep in adjustment. Lapicque's clinical chronaximeter uses keys in the circuit, as does Lucas', which are a source of considerable mechanical trouble. For these reasons we have devised, with the assistance of Professor Lindley Pyle, professor of physics at Washington University, an apparatus which we feel does away with these objections.

DESCRIPTION OF INSTRUMENT BY PROFESSOR LINDLEY PYLE

The problem was to incorporate into an electrical circuit an apparatus which could be opened and closed under the control of the operator and permit the flow of a current for a definite and measurable interval of time, approximately only a few ten thousandths of a second. The apparatus was to be so adjustable that the time interval of current flow could be increased by steps up to the order of a hundredth of a second. It was to be so reliable and constant in action that any chosen current pulse could be repeated as to duration of flow at any time the operator required it. Furthermore, the apparatus was to be light and portable yet sufficiently robust to insure permanency in its time constants. For this reason the method of the heavy topped pendulum as used in the Lucas pendulum was discarded and recourse was had to a spring driven device. After several weeks of experimentation the apparatus pictured in Figure 1 was found to fulfil the required conditions.

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14. Adrian, E. D.: The Electrical Reactions of Muscles Before and After Nerve Injury, Brain **39**:1, 1916; The Recovery of Conductivity and of Excitability in Nerve, J. Physiol. **50**:345 (Sept.) 1916; Physiological Basis of Electrical Tests in Peripheral Nerve Injury, Arch. Radiol. & Electroth. **21**:379 (May) 1917; Conduction in Peripheral Nerve and in the Central Nervous System, Brain **41**:23 (June) 1918; Response of Human Sensory Nerves to Currents of Short Duration, J. Physiol. **53**:70 (Sept.) 1919.

A light aluminum wheel approximately 8 inches in diameter is mounted on a ball-bearing shaft fastened to a bed-plate of brass 3/16 inch in thickness. One end of a helical coil of steel wire is secured to the bed-plate, the other end being fastened to the wheel. The fundamental idea is to wind up the spring by a rotation of the wheel to a definite position. The wheel, on being released, acquires a high peripheral speed and then automatically brings into action a sliding contact of an attached metal brush across a stationary copper plate of variable width mounted close to the wheel rim. A current flows during the time of contact.

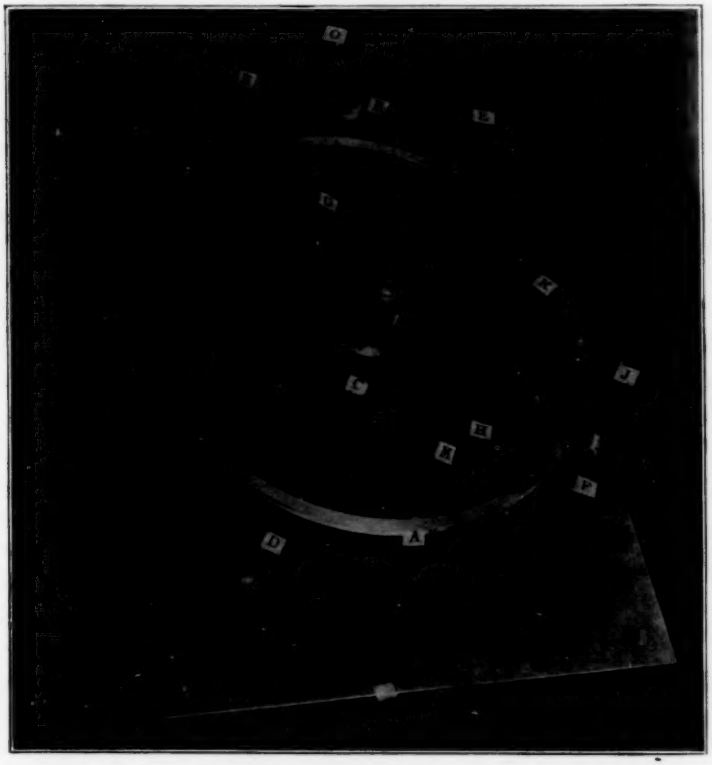

Fig. 1.—Chronomyometer.

Referring to Figure 1, the knob *M* is grasped by the hand and the wheel is rotated right-handedly through about one and a half revolutions. A trigger *D* is then raised to engage with a pin *K* mounted on the lower side of the wheel rim. The mechanism is now "set," ready for operation, and awaits only the depression of the trigger, *D*. A piece of spring-brass wire is mounted on the periphery of the wheel as shown at *A* and constitutes the brush referred to. The outer angle of the wire brush is faced with a strip of platinum soldered to the wire. The

block *E* is made of nonconducting red fiber composition so shaped as to have a curved surface concentric with the axis of the wheel. Embedded in this fiber and inlaid so as to be flush with its surface is a piece of heavy-gage copper, *B*, cut into five "steps," or widths, as indicated. A binding post, *O*, is in metallic connection with the copper plate, *B*, by means of a screw penetrating the fiber block. A second binding screw (not shown) is soldered to the brass bed-plate. The fiber block is supported on two brass rods that run through two holes in the bed-plate. A set-screw at *N* may be clamped against one of the support rods so that the fiber block may be set at any desired position. A leather-faced brake-shoe is held away from the outer face of the wheel by a trigger *F*.

The mechanism being set for action and included in the desired electrical circuit by connections to its binding posts, the trigger *D* is pressed and the wheel gathers speed under the action of the stressed spring *C*. The spring-wire brush, *A*, ultimately rides up upon the fiber block, *E*, and, dragging along its smooth surface under a firm pressure, it crosses the copper plate, *B*, at high speed closing the electrical circuit for a definite interval of time. Just after *A* has broken contact with *B* a strip of brass swinging from a pivot mounted on a wheel-spoke at *G* strikes *H* and disengages the trigger *F*. A powerful spring attached to *J* throws the brake-shoe into contact with the periphery of the wheel and brings it to rest before the inertia of the wheel puts a backward twist into the spring, *C*, and before the brush, *A*, passes *B* the second time.

It was found necessary to give the brush at least a 4-inch travel along the fiber block before it crossed the plate *B* in order that any vibration and chattering of the brush due to the initial impact of the brush with *E* might be damped out before the closing of the circuit occurred—otherwise the contact was uncertain. It is desirable to wipe the rubbing surfaces of *B* and *A* and *E* with a piece of chamois to remove dust before each trial. At frequent intervals the faces of *A* and *B* should be wiped with a piece of chamois impregnated with the finest emery dust. With these precautions one may feel assured that at any time a current pulse may be reproduced of unvarying time lapse for any chosen position of the contact plate, *B*.

The duration of contact for each of the five widths of *B* were obtained by an electrical method. A nonabsorbing mica condenser of known capacity was charged through a known resistance by a long-time contact with a battery. The accumulated charge was discharged through a ballistic galvanometer, and the throw noted. With the same circuit constants the condenser was given a short-time charge through the contact-maker under discussion and the ballistic throw corresponding

to the short-time charge noted. By a formula known to physicists the duration of the short-time contact was calculated.

The duration of contact for each of the widths of plate were found to be 0.00015 second, 0.00033 second, 0.00065 second, 0.0008 second and 0.009 second. From these figures it is readily determined that each 1 mm. width of copper plate produces a contact approximately 0.000082 second in length. It is possible to secure any value of current duration desired by having a series of copper plates, *B*, of varying widths.

The instrument described we have called a "chronomyometer" because it enables one to measure the time of flow of a current necessary to cause a muscle to contract whether the nerve supply is intact or degenerated.

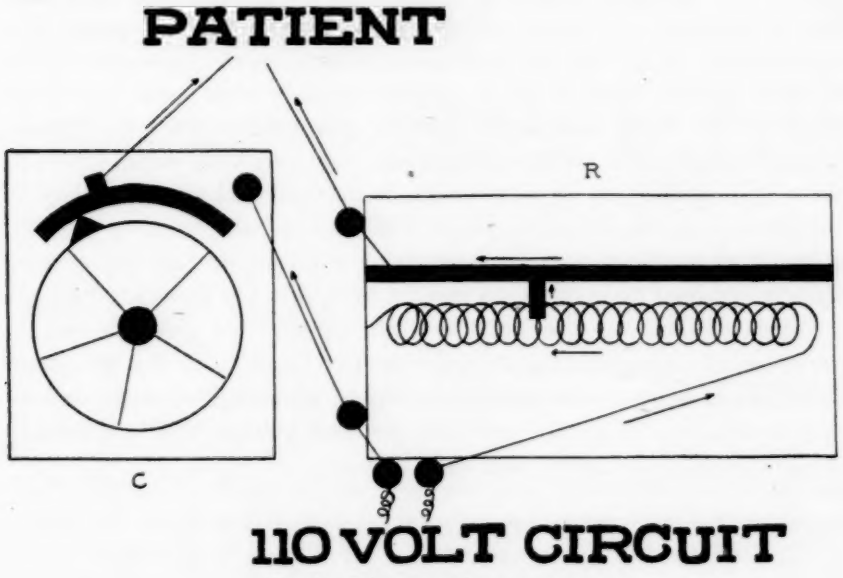

Fig. 2.—Diagram of apparatus.

Prolonged use seems to show that the device is consistent in its action and that any possible variations in the elastic properties of the driving spring are inappreciable.

Figure 2 is a diagram of the electrical apparatus and connections when in use. The chronomyometer, *C*, is in series in the circuit so that the current is flowing only when the brush, *A*, of Figure 1 is in contact with the plate *B* of Figure 1. A resistance coil, *R*, is also placed in the circuit in series so that the voltage of the current used could be varied from 0 to 110 volts. The amperage varies between 6 and 8 milliamperes. A direct current must always be used, and if only an alternating current is available, the direct current may be generated by a small direct current dynamo which is driven by an alternating current motor.

In testing a patient a large indifferent electrode is connected to the positive pole (anode) and an ordinary 1 cm. diameter stimulating electrode to the negative pole (cathode).

The arbitrary values of the duration of the current which the chronomyometer delivers were selected because we felt that any shorter steps would be of no clinical value and would make the procedure too complicated. A nerve-muscle-complex of 0.0008 second was selected as the longest for it was felt that in exceptional cases, at least, a current of longer duration might fall within the limits of a rapid muscle complex and thus lead to false conclusions.

The method of testing is briefly illustrated by the following example: Suppose we want to test a gastrocnemius that has no voluntary movement. The indifferent electrode is placed on the thigh on the same side, the stimulating electrode on the gastrocnemius motor point, the apparatus is set so that the duration of the current corresponds to the longest nerve-muscle complex, 0.0008 second; start with a voltage of about 30. If no contraction follows, gradually increase the current strength until a contraction is obtained. If we obtain none with 110 volts we consider no nervous tissue is present, and the procedure is repeated using the muscle complex 0.009 second and starting with 10 volts; if this fails to cause a contraction a longer duration of current may be obtained by making the contact by hand. On the other hand, if a contraction follows stimulation with the current that gives the longest nerve-muscle complex, the duration is shortened until the minimum duration, no matter how strong the current, which will cause a contraction is obtained. We also determine the least voltage that is necessary to get a contraction at this minimum duration.

PHYSIOLOGIC PRINCIPLES OF MUSCLE AND NERVE-MUSCLE RELATIONS BASED ON A CONSIDERATION OF THE LITERATURE

The more recent investigations on the properties and electrical phenomena exhibited by muscles and nerves have been carried on primarily by Keith Lucas,[12] Lapicque,[13] Sanderson,[15] Adrian,[14] Gotch,[16] and A. V. Hill.[17] To understand this work properly a clear conception

15. Sanderson, J. Burdon: The Electrical Response to Stimulation of Muscle, and Its Relation to Mechanical Response, J. Physiol. **18**:117, 1895.

16. Gotch, F., and Burch, G. J.: The Electrical Response of Nerve to Two Stimuli, J. Physiol. **24**:410, 1899; The Effect of Local Injury Upon the Excitatory Electrical Response of Nerve, ibid. **28**:32; The Submaximal Electrical Response of Nerve to a Single Stmulus, ibid. p. 395; Gotch, F.: The Delay of the Electric Response of Nerve to a Second Stimulus, ibid. **40**:250, 1910.

17. Hill, A. V.: A New Mathematical Treatment of Changes of Ionic Concentration in Muscle and Nerve Under the Action of Electric Currents, with a Theory as to Their Mode of Excitation, J. Physiol. **40**:190, 1910.

is necessary of the excitation time of nerves and muscles, the term applied to it by Lucas.[12]

The excitation time is based on two factors: (1) the minimal strength, and (2) the minimal duration of a constant current, that is, a current which reaches a certain voltage immediately and maintains this voltage as long as it is flowing, in contradistinction to a condenser discharge whose current gradually decreases in intensity. It is also important to realize that constant or galvanic currents and faradic currents are the same kind of electricity, faradic differing from galvanic only in that it flows intermittently for short periods of time.

If one stimulates a nerve muscle preparation with a constant current it is found that the contraction of the muscle is dependent not only on

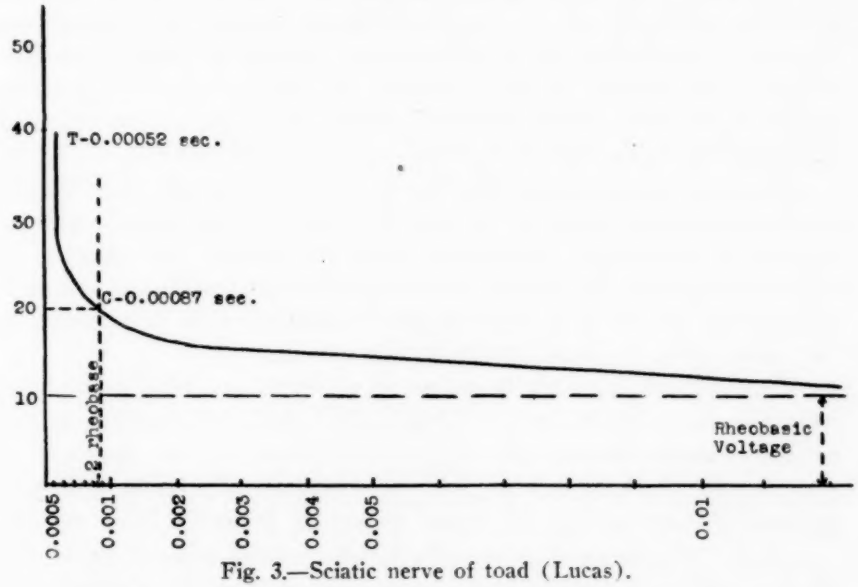

Fig. 3.—Sciatic nerve of toad (Lucas).

the strength of the current, but also on its duration. No matter how strong the current is, that is the voltage, the current must flow a certain length of time in order to get a contraction, and if this is shortened no reaction follows. Conversely, no matter how long the current flows, if the current is not more than a certain strength, no contraction of the muscle will be obtained. Between these extremes as the duration of the current is decreased the strength of the current must be increased to produce a contraction, and vice versa, the duration has to be increased as the strength is decreased. In order to understand the relationship between these two factors, the minimum strength and the minimum duration must be determined in each case. Lucas and Adrian have shown this graphically both in a toad's muscle (Fig. 3)

and in a human tibialis anticus with intact nerve supply (Fig. 4). The ordinates show the strength of current, the abscissae the duration of the current. The minimum duration of a current necessary to stimulate a muscle without nerve supply is much longer than that necessary to stimulate a muscle with an intact nerve (Fig. 5).

An analysis of these curves shows that at long, or, as they called it, "infinite" duration there is a minimum strength below which no contractions occur, and as the duration is decreased the strength must be increased until a point is reached where, no matter how strong the current, no contractions follow. This minimum of duration is the measure of the excitation time. The minimal strength current of infinite duration necessary to get a contraction, Lapicque [13] called rheobasic voltage. He noted that a sudden change in the curve occurred approximately at a point at which twice the rheobasic voltage was used and the length of time the current flowed with this strength he called chronaxie. Adrian [14] has found that the chronaxie of a human muscle with intact nerve supply was about 0.00016 second while the chronaxie of a muscle whose nerve had degenerated was about 0.01 second.[18]

We want to emphasize that the chronaxie is not the same as the absolute minimum duration of current required to produce a contraction. In a muscle with intact nerve these two points differ slightly, as shown in Figure 4, the difference being 0.00010 and 0.00016 of a second, while when the nerve is degenerated the difference in these points is very great (Fig. 5), 0.003 and 0.01 of a second.

The difference between the chronaxie of nerves and muscles explains why a muscle whose nerve is degenerated does not respond to the ordinary faradic current but only to the galvanic, for the duration of each faradic shock is shorter than the chronaxie of muscle while the galvanic current is always much longer in duration than muscle chronaxie. The repeated shocks of a faradic current make it impossible for the patient to endure a strong enough current to stimulate the muscle when its nerve supply is impaired, and thus it is of limited clinical value.

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18. The length of the chronaxie of normal muscles and muscles without nerve supply recorded by Lapicque in his first report (Techniques nouvelles pour l'electro-diagnostic, Compt. rend. Acad. d. Sc. **161**:643 [Nov. 22] 1915) agrees very well with Adrian's (The Electrical Reactions of Muscles Before and After Nerve Injury, Brain **39**:1, 1916. Physiological Basis of Electrical Tests in Peripheral Nerve Injury, Arch. Radiol. & Electroth. **21**:379 [May] 1917), but in his second report (Presentation d'un chronaximétre clinique, Compt. rend. Soc. de biol. **78**:695, 1915) it is about ten times as long. As our results correspond to those of Adrian's we feel that his figures are more nearly correct. The difference in Lapicque's figures may be due to the calibration of his chronaximeter.

CLINICAL APPLICATION OF CHRONAXIE

Adrian [14] has used the Lucas pendulum with interrupting keys to vary the duration of the current and a potentiometer to vary the strength of the current. With this apparatus he has determined the curve for a muscle with intact nerve supply (Fig. 4), a muscle without nerve supply (Fig. 5), and a muscle with incomplete division of its nerve (Fig. 6). It will be noted that in Figure 6 the curve has two phases; in reality, therefore, it is fair to assume that there are two separate curves as indicated by the dotted lines; one of these represents the strength-duration curve of a muscle without nerve supply with a chronaxie of 0.004 second (curve A) while the other represents the strength-duration curve of a muscle with intact nerve supply with a

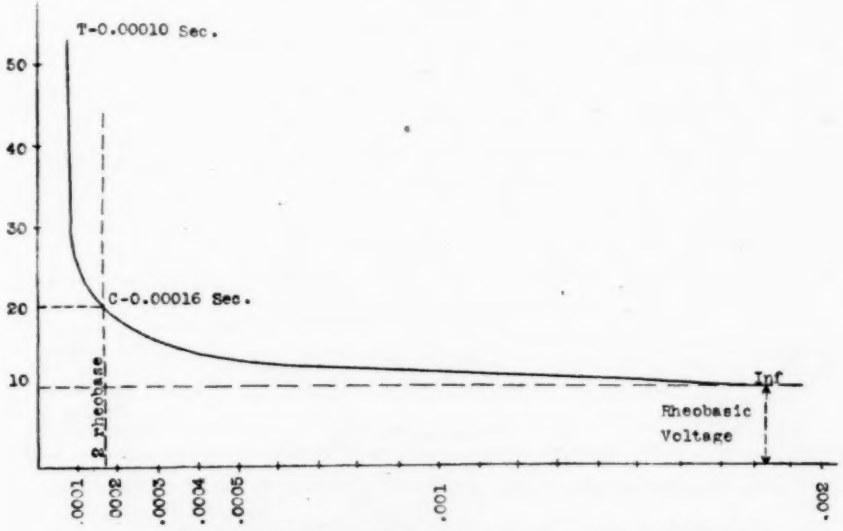

Fig. 4.—Human tibialis anticus, intact nerve supply (Adrian).

chronaxie of 0.0005 second (curve B). This means that if we consider chronaxie as the measure of the excitation time of the tissue we would conclude that 0.004 second meant that only muscle fibers were present. If, however, the true minimum duration of the current is used as the measure of the excitation time of the tissue, it will be seen to be less than 0.0005 second and this would mean that some nerve tissue is present. *We feel that the true minimum duration of the current, no matter how strong the current, is the real measure of the excitation time of the tissues in question and we therefore have disregarded arbitrary chronaxie in our tests.* Cases 4 and 10 illustrate this very well.

CASE 4.—On September 18 the rheobasic voltage was 20 V, the chronaxie at 40 V, twice rheobase, was 0.009 second, and thus one would

conclude that there was no nerve tissue present. The minimum duration, however, was 0.00033 second with 100 V, and thus we concluded that some nerve fibers were present in the muscles tested and gave the patient a favorable prognosis. The ultimate recovery of voluntary power in these muscles bears out our opinion.

In order to avoid confusion we prefer to use the terms "muscle complex" and "nerve-muscle complex." By muscle complex we mean that the minimum duration of the current necessary to stimulate a muscle falls within the range of time determined clinically and experimentally for muscles without their nerve supply, 0.02-0.004 second; by the term "nerve-muscle complex" we mean that the minimum duration of the current necessary to stimulate a muscle falls within

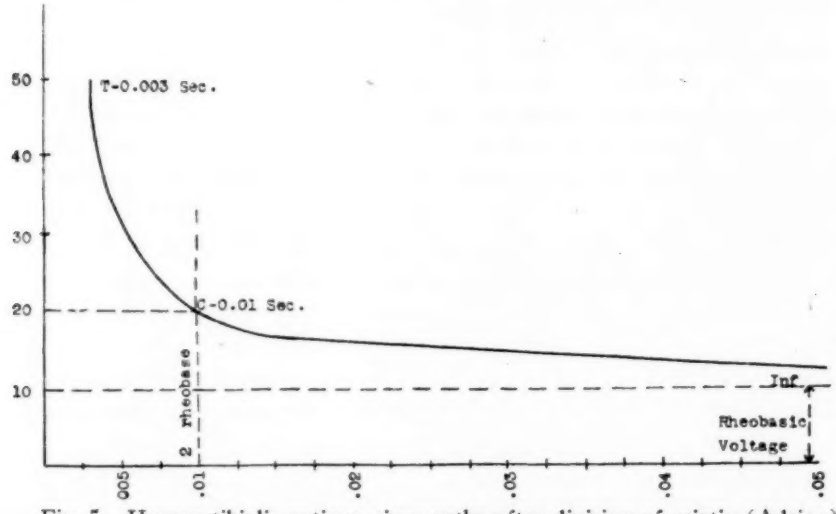

Fig. 5.—Human tibialis anticus, six months after division of sciatic (Adrian).

the range of time determined clinically and experimentally for muscles with intact nerve supply, 0.0009-0.00008 second. The terms long, medium and short are added to indicate whether the nerve-muscle complex is normal or approaching normal, the short response being the normal.

From the three factors, the type of contraction, the minimum strength of current and the minimum duration of the current, the degree of impairment of the nerve supply and the condition of the muscles are determined, and by repeating these tests at varying intervals the progress of the case may be followed accurately. The changes that occur in these cases may be observed by a study of our protocols.

Before considering our own results it will be necessary to point out what we believe to be the objections to previous methods that have been used for testing nerves and muscles.

DISCUSSION OF OTHER METHODS

Chronaxie.—Adrian [14] considers chronaxie the important criterion of the condition of the tissues but also insists that the full curve should be determined in each instance so that a two phase curve, as shown in Fig. 6, will not be overlooked.

This method has the practical disadvantage, however, of taking hours to make these determinations in cases in which a number of muscles are involved and besides, we believe, the determination of the complete strength duration curve is unnecessary.

He, however, also makes this statement: [14] "A current of 0.004 second will excite nerve fibers if it is strong enough, but it will not

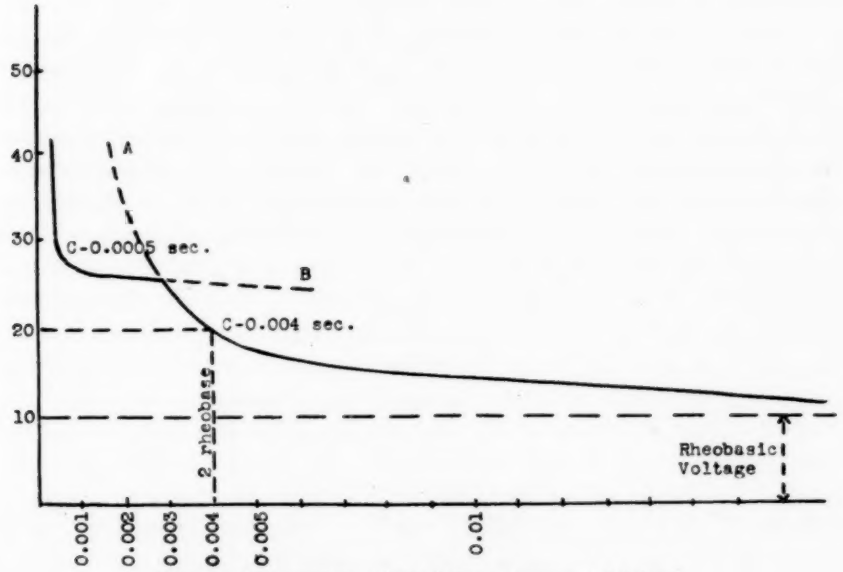

Fig. 6.—Incomplete sciatic injury, human (Adrian).

stimulate muscle fibers, however strong it may be. So if we could always use a current of this duration and of variable strength there would be no need to determine the full curve." Such a method would be essentially what is done with a faradic current unless Adrian means to use only a single shock instead of the repeated shocks of a faradic current but even then a change in the nerve muscle complex could only be detected by the variation in the minimum strength of current used.

According to Adrian the important criterion of regeneration is the decreasing strength of current necessary to bring out nerve muscle chronaxie after it has once begun to appear. As will be seen later, we have found that the change in the minimum duration of the current is the most important criterion.

The minimal strength of current will vary with the tissue resistance, temperature of the muscle, etc., even if there were no change in the innervation of the muscle tested. On account of this great variation in tissue resistance the minimum duration of the current and not the minimum strength of the current must be used as the measure of the excitation time, and consequently the determination of the full strength-duration curve is unnecessary.

The objections to chronaxie determinations are that they do not give the true minimum duration and thus facts are disregarded that we believe are of vital importance in determining the regeneration of a nerve. Also true nerve-muscle chronaxie has seldom been observed in injured nerves until regeneration is nearly complete and at that time voluntary movement has begun to appear.

Condensers.—Condenser systems of the Lewis Jones [19] type enable one to test a muscle with currents of different duration, but Jones [19] himself points out that if the current strength is altered the time of discharge of a given condenser is also altered. Hernaman-Johnson [20] has devised an apparatus to change the voltage of Jones condensers, but this does not regulate the time of discharge which is affected by the voltage. The time of discharge for different voltages could be calculated, but for clinical purposes this is impracticable. Furthermore, a condenser of known capacity charged with a constant voltage discharges in a definite time, but as it discharges the intensity of its current decreases gradually to zero. Roberts [21] has pointed out that a portion of such a discharge is below the minimum strength of a current, at infinite duration, necessary for stimulation, and this portion is ineffective. Since the resistance of tissues varies greatly at all times, the size of the minimum strength current necessary to stimulate a muscle will vary. It follows, therefore, that the amount of current which is below the minimum strength will vary with this resistance, and the duration of the effective part of a condenser discharge varies greatly even though a given condenser is charged with the same voltage. For this reason the value of the constant current becomes apparent for it attains its maximum voltage at once, and this is maintained throughout the period of flow.

For these reasons it is obvious that the results obtained with condenser discharges when compared to tests on normal muscles or previous tests on any muscle are unreliable.

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19. Jones, H. L.: The Use of Condenser Discharges in Electrical Testing, Proc. Roy. Soc. of Med. **6**:49 (Jan.) 1913; Medical Electricity, Ed. 6, London, 1913.

20. Hernaman-Johnson: Use of Condensers in Diagnosis, Prognosis and Treatment of Muscles Following Nerve Injuries, Lancet **1**:396, 1916.

21. Roberts, F.: Degeneration of Muscle Following Nerve Injury, Brain **39**: 297, 1916.

Galvanism and Faradism.—The objections to this method of testing muscles electrically have been pointed out by many observers and may be summarized as follows: First, faradism usually returns at about the same time that voluntary movements return, and therefore is of little clinical value. This fact does not appear to be in harmony with the previously stated physiologic facts, but this discrepancy is explained by the fact that the faradic current is a repeated series of shocks, each one having the duration of a long nerve-muscle complex. In cases of regenerating nerves before voluntary movement has returned we shall see that it is necessary to use currents of relatively strong voltage to produce contractions. When this voltage is used with an induction coil, the pain is so great that it is unbearable and in addition the current spreads to surrounding healthy muscles rendering conclusions difficult or impossible. Second, the duration of the faradic shock cannot be varied and would be of no value in showing the progress of a case even if a response could be elicited before voluntary contractions returned. Third, the comparison of the response to negative and positive threshold galvanic currents varies in normal muscles, and when strong currents are used it is difficult to distinguish between the return to normal of stimulated antagonist or neighboring muscles, and the slow contraction of the muscles being tested. The threshold strength varies several milliamperes if exactly the same motor point is not stimulated each time. This renders tests from month to month of little value. Fourth, the entire procedure is time consuming, requires considerable experience and is difficult to interpret.

RELATION OF INDUCED WAVES OF OPENING AND CLOSING (LAUGIER [22])

This method supplies a means of testing a muscle with varying strength of current but only the duration of one muscle complex and one nerve-muscle complex. These values are dependent on the individual coil employed, and a ballistic galvanometer is necessary to determine the strength of current used. The results must be expressed in relative figures derived by dividing the current used in the closing wave by the current used in the opening wave. Objections to this method are: First, the variable is the strength of current which is dependent on many other factors than the condition of the muscle in question; second, the duration of the current cannot be varied; third, the method is technically difficult to carry out; and fourth, the apparatus is not readily transported.

EXPERIMENTAL DATA

In the course of some experiments on methods of repair of peripheral nerves in dogs, the muscles innervated by these repaired nerves were

22. Laugier: Biol. méd. 1914, p. 89.

tested electrically with our chronomyometer. Other experiments were carried out in order to test the changes in the nerve muscle complex during degeneration and regeneration of an injured nerve. The nerves were exposed under ether anesthesia from time to time under strictest aseptic precautions, and thus by direct stimulation of the nerve trunks with the faradic current we determined when regeneration had occurred and the results compared with the chronomyometer findings. Nerves were excised from time to time in order to check up by histologic studies the stage of regeneration of the nerves. All specimens were stained by the Silver-Pyridin method described by Ranson.[3] In all we have done eight nerve sutures, and fifty observations were made on them. (The factor of pain was entirely eliminated by the use of ether anesthesia.)

A study of the tables and protocols of our experiments reveals the following facts:

The nerve muscle complex remains normal for a period varying from forty-six to seventy hours after cutting the nerve trunk (Exper. 89, 91, 93, 113 and 121). In no case was a nerve-muscle complex found on a dog after seventy-five hours.

When the irritability of the nerve begins to diminish it changes rapidly and within from two to five hours only the muscle complex is present. In Exper. 93 the nerve-muscle complex did not begin to disappear until some time between the fiftieth and sixty-eighth hour, but then in one hour and forty minutes it changed from 0.00033 second to 0.00065 second, to 0.0008 second, and then to 0.009 second, the muscle complex. Throughout these tests the same voltage was always used.

As regeneration occurs the slow nerve muscle complex appears first, and then gradually the nerve-muscle complex shortens until it finally becomes normal, 0.00015 second. In these experiments the animals were anesthetized so that the factor of pain could be eliminated and the strength of the current could be increased to the point at which the current was seen to spread to normal muscles.

Faradic response was only elicited when the nerve muscle complex was normal. It could not be elicited earlier even under anesthesia, for when currents as strong as those used with the chronomyometer were used with faradic stimulation, the current spread to normal muscles so that accurate observations were impossible. Whenever the nerve-muscle complex begins to appear it is an evidence of return of regeneration. This we have checked up by histologic examination and by direct stimulation of the nerve trunk (Exper. 51, 81, 91, 99, 113 and 121).

EXPER. 51.—Double lateral anastomosis of peroneal nerve to tibial nerve, tested 221 days after operation and nerve-muscle response 0.00015 second in muscles of peroneal and tibial distribution. Central tibial nerve cut and tested eighteen days later, showed 0.00015 second over peroneal nerve distribution and 0.009 over tibial nerve (with and without anesthesia). Direct stimulation of nerve trunks gave no tibial nerve response but normal peroneal response. Microscopic sections of tibial nerve showed no axis cylinders.

Summary: Nerve-muscle complex before cutting, muscle complex eighteen days after, confirmed by direct stimulation of nerve trunk and histologic examination.

EXPER. 81.—Double lateral anastomosis of peroneal nerve to tibial nerve, tested 106 days later. Reaction: 0.00015 second in anterior and posterior muscles of leg though those innervated by peroneal nerve very weak. Direct stimulation of nerve trunks gave active response of those innervated by tibial nerve but very weak over peroneal nerve distribution. Microscopically there were fewer axis cylinders in the peroneal nerve than in the tibial nerve.

Summary: Weak muscular response to 0.00015 second checked by direct stimulation of nerve trunk and microscopically.

EXPER. 93.—Left sciatic cut and resutured. Nerve-muscle complex 0.00015 second before cutting and for 50 hours after, 0.00033 second 68 hours after operation, 0.00065 second 50 minutes later, 0.0008 second 20 minutes later, 0.009 second 30 minutes later. The voltage was the same in all tests.

Summary: Nerve-muscle complex normal 50 hours after cutting, almost normal 68 hours after cutting but 1 hour 40 minutes later only muscle complex present, that is, irritability of nerve normal for 68 hours after cutting but irritability lost rapidly when it started to decrease.

EXPER. 99.—Nerve tested three months after regeneration and 0.00015 second response found, confirmed by direct stimulation of nerve trunk.

EXPER. 113 and 121.—Opened three days after cutting the nerve, and direct stimulation of its trunk failed to cause muscular contractions. Only muscle complex present. Histologically beginning degeneration.

Summary: Muscle complex after three days; no response to stimulation of nerve trunk and beginning degeneration histologically.

EXPER. 89.—Left sciatic cut and resutured—motor points of left leg tested in each case. Details of this experiment are given in Table 1.

EXPER. 91.—Left sciatic cut and resutured—motor points of left leg tested each time. The details are given in Table 2.

CLINICAL CASES

In the past year about thirty-five cases of peripheral nerve lesions have been tested with the chronomyometer. Many of these patients have been followed with frequent examinations until recovery was complete. Others have not completely recovered as yet, while some have either not returned for examination or the examination was not considered of value, as in cases in which the lesion involved the central neuron.

In each case the following points were studied: sensory changes, trophic disturbances, voluntary control of muscles, functional disability

and electrical tests (the chronomyometer was used in all cases and faradism in many). We also looked for Tinel's sign and the pinching test of Athanassio Bénisty. Of the thirty-five cases examined, the

TABLE 1.—DETAILS OF EXPERIMENT 89

Date and Time of Examination	Duration of Current in Seconds	Approximate Strength of Current in Volts	Faradism
6/28/20 Before operation	0.00015	..	+
45 minutes later	0.00015		
2½ hours later	0.00015	60	
4 hours later	0.00015		
9 hours later	0.00015		
24 hours later	0.00015	..	+
46 hours later	0.009	..	—
7/ 8/20	0.009	..	—
7/22/20	0.009	..	—
8/21/20	0.009	..	—
9/ 3/20	{0.0008	21	—
	?0.00006?	42	—
9/13/20	{Posterior muscles 0.00033	42	—
	?Anterior muscles 0.00015	42	—
9/20/20	{0.00083	22	?
	?0.00015	28	
10/11/20	0.00015	16	+ but very weak
10/18/20	0.00015	16	+ with good contractions

Summary: Left sciatic cut and normal nerve-muscle response for at least twenty-four hours and only muscle complex after forty-six hours. First nerve-muscle complex sixty-seven days after operation and duration of current gradually shortened to normal in thirty-eight days. Faradism was weakly present at this time, therefore chronomyometer test showed regeneration thirty-eight days ahead of faradism. Regeneration confirmed histologically.

TABLE 2.—DETAILS OF EXPERIMENT 91

Date and Time of Examination	Duration of Current in Seconds	Approximate Strength of Current in Volts	Faradism
6/28/20 Before operation	0.00015	57	+
½ hour after operation	0.00015	57	+
20 hours later	0.00015		
50 hours later	0.00015		
68 hours later	0.00015		
69 hours later	0.00015		
70 hours later	0.00015		
75 hours later	0.009		
7/ 8	0.009		
7/22	0.009	..	—
8/21	0.009		
9/ 3/20	0.009		
9/13	0.009	..	—
9/20	0.009	..	—
10/11	{0.00033 doubtful at	33	
	?0.00083 definite at	55	—
10/18	0.00015	42	—
10/26	0.00015	22	± anterior muscles; + posterior muscles
11/ 6	0.00015	14	+

Summary: Nerve-muscle complex lost between seventy to seventy-five hours began to return after from eighty-two to one hundred and two days and completely returned after one hundred and nine days, while faradism first returned after one hundred and seventeen days. Regeneration was confirmed microscopically.

protocols of only twenty-one are recorded as the others threw no light on this investigation. We record here only the positive findings in each of these cases.

A study of the twenty-one cases reported and Table 3 lead us to the following conclusions:

1. As regeneration occurs the long nerve-muscle complex appears first and as regeneration continues the nerve-muscle complex shortens until finally it reaches the value of a normal nerve-muscle complex. The early appearance of a long nerve-muscle complex enabled us to recognize that there was beginning regeneration. Thus an earlier diagnosis and an earlier favorable prognosis was possible. The ultimate recovery of all patients in whom this was observed has confirmed this opinion.

The minimum voltage necessary to cause contractions may remain practically the same at each test although the duration shortens, but in some instances it may vary considerably from time to time and this is due to the variation in tissue edema, temperature of the muscles, etc.

TABLE 3.—TIME CHRONOMYOMETER RESPONSE PRECEDED OTHER SIGNS OF REGENERATION

Case No.	Diagnosis	Before Faradism	Before Voluntary Contractions	Before Decrease in Anesthesia	Remarks
1	Left sciatic paralysis	3 months	3 months	2 months	
2	Incomplete brachial plexus injury	3 months	3 months	5 months	
3	Right sciatic incomplete	4 months	2 months	2-4 months	
4	Left sciatic paralysis	6 months	1-2 months	No gain	
5	Poliomyelitis	Not tested	2 months		
6	Bell's palsy	No gain	23 days	.....	No degeneration of nerve
7	Left facial paralysis	Not tested	1-3 months		
8	Left facial paralysis	1 month	23 days		
9	Bell's palsy	Not tested	1 week	.....	No degeneration of nerve
10	Right facial paralysis	Probably 3 months	Probably 3 months		
11	Left facial paralysis	Not tested	1 week	.....	No degeneration of nerve
12	General polyneuritis	Faradism too painful			

In Table 3 it will be noted that the nerve-muscle complex returned from one to six months before faradism, one week to three months before voluntary contractions appeared and from zero to five months before anesthesia decreased. The greatest gain was observed in cases of injury to the long nerves and the least gain in the facial nerve lesions. This is, of course, to be expected for the distance the regenerating axis cylinders must grow and the size of the muscles innervated influences markedly the time between beginning and complete recovery. The time between degeneration and regeneration of the nerve determines to a large extent the amount of atrophy of the muscles, no matter how well they are cared for.

2. The nerve-muscle complex had disappeared by the fifth day in the posterior group of muscles in Case 3. This was the only case we saw

early enough to enable us to determine when the nerve-muscle complex first disappeared. This agrees with our animal experiments in which the nerve-muscle complex disappeared invariably at the end of seventy-six hours after complete severance of the nerve.

Adrian has reported a case of facial paralysis in which the nerve muscle chronaxie disappeared between the fifteenth and sixteenth day. This would indicate that in human nerves there is considerable variation as to when the nerve muscle complex is lost.

3. The presence of the nerve-muscle complex in cases in which there is no voluntary movement is a valuable prognostic sign as illustrated by Cases 6, 9 and 11. In these cases because of the normal nerve muscle complex we gave a good prognosis for early recovery. Therefore, whenever the nerve-muscle complex is present, even if there is no voluntary movement, no surgical interference should be considered.

4. Faradic response did not return until the nerve-muscle complex was 0.00033 second or shorter, that is, not until the irritability of the nervous tissue reached normal limits and voluntary movements were beginning to return. Case 12, in which hyperalgesia was present, illustrates strikingly the difficulty of testing with faradism on account of the pain, for even with weak currents the repeated stimulus is painful while a single shock of shorter duration is well tolerated by the patient.

5. Voluntary contractions returned at about the same time that faradism returned or when the nerve-muscle complex had reached normal limits.

6. The decrease in the area of anesthesia may occur parallel with the appearance and shortening of the nerve-muscle complex or it may lag considerably behind. In cases of motor nerve lesions, as in the facial, this part of the examination is, of course, useless, and conversely the electrical test at first seems to be useless in lesions of sensory nerves. That this is probably not true is suggested by Adrian's [14] work in which he determined the strength-duration curve for sensory nerves. This suggests the same possibilities for sensory nerves as we have found for motor nerves although we have made no observations on this point.

7. In incomplete nerve lesions, as in Case 3, in which only a few fibers remain intact, the detection of one muscle showing even the slowest nerve-muscle complex may be of great diagnostic and prognostic value In this case it saved the patient an exploratory operation and recovery followed in due time.

8. In extensive lesions as those of a plexus, Case 2, or a high sciatic lesion, Case 17, the progressive lengthening of the nerve-muscle complex as one proceeds distally from the lesion may mean one of two things. If the case is seen early it means that the injury is incomplete,

while if the case is seen late it means either that the injury is incomplete or that regeneration has begun. The latter point is easily determined by a subsequent examination. In either case we feel that the prognosis is usually good and that operation is not indicated until it has been proved that regeneration is stationary after repeated examinations.

9. The only case of poliomyelitis cited, Case 5, shows a change from the muscle complex to the normal nerve-muscle complex in two months in some of the muscles, which we interpret as meaning that the anterior horn cells were regaining their activity. This method offers a possible means of determining with mathematical accuracy when the process of recovery in the affected nerves in poliomyelitis has come to a standstill.

10. When patients were seen a few months after injury and showed complete paralysis of a given nerve we delayed making a prognosis or stating whether operation was advisable until the axis cylinders might have had time to reach the nearest muscle distal to the lesion. We used as a basis for estimating when this time should be the measuring of distance from the lesion to the motor point of the nearest muscle. This distance in centimeters gives the number of weeks it should take the regenerating axis cylinders (if regenerating) to reach the nearest muscles and thus be detected by the electrical test. One cm. per week seems to be a conservative average rate of regeneration, but it must be used with judgment for each case varies and the entire picture must be considered. Cases 1 and 4 show the value of waiting in old cases. We feel, however, that a case seen early, unless the injury is only a few centimeters from a motor point, is entitled to an exploratory operation if no signs of recovery are present at the end of from six to eight weeks.

11. We have some evidence to indicate that there is some relation between the number of axis cylinders in a nerve and the length of the nerve-muscle complex. In Case 3 we were able to study the popliteal nerve in cross section and found that 5 cm. below the knee, axis cylinders were present in very large numbers (appeared like a normal nerve) while 5 cm. distally from that point the number of axis cylinders was definitely decreased. This decrease continued until at the ankle there were about one or two axis cylinders to a low power field. The electrical response of the gastrocnemius was shorter than that of the tibialis anticus, and as the former muscle gets its innervation higher up the variation in the length of the nerve-muscle complex may have been dependent on the number of axis cylinders present. It may of course be possible that the variation in the threshold duration necessary to produce a contraction accounts for the difference in electrical reaction at different stages of regeneration. Further observations are necessary to clear up this point.

CASE 1.—B. S., bullet wound in thigh January, 1920, tourniquet applied on lower third of thigh for two days to control hemorrhage, followed by paralysis of left leg.

TABLE 4.—FINDINGS IN CASE 1

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Anesthesia to Pin Prick	Remarks
7/10/20	All of left leg	0.009	85	Fair	None	That of sciatic lesion	Trophic ulcer of heel and great toe; massage and electricity started; impression-degeneration of sciatic nerve below thigh
	All of right leg and left thigh	0.00033	82	Good	Normal	None	
8/24/20	Soleus and median head of gastrocnemius	0.00066	90	Weak	None	Same as on 7/10	Muscles somewhat spastic; good prognosis given; faradism negative
	Tibialis anterior	0.0008	90	Weak	None	Same as on 7/10	
10/20/20	Soleus and median head of gastrocnemius	0.00033	105	Weak	None	To mid-leg lateral and below malleolus medial	Edema of leg, infection of toes; no massage for two months; faradism negative
	Tibialis anterior	0.00015	88	Weak	None		
11/30/20	Anterior and posterior muscles of left leg	0.00015	95	Fair	Slight	Sole of foot and one inch up all around foot	Faradism weak; infection improving

Summary—Diagnosis: Complete degeneration of sciatic nerve below thigh but prognosis delayed to allow regenerating fibers (if present) to reach muscles and skin of leg. Eight months after injury long nerve-muscle complex first appeared and good prognosis given. Three months later nerve-muscle complex was normal, slight voluntary movements were present and anesthesia was greatly reduced. Gain of three months over voluntary movements and faradism and two months over sensory. (In another similar sciatic lesion the sensory paralleled the nerve-muscle response.)

CASE 2.—H. brachial plexus injury received November, 1918, explored [illegible]

CASE 2.—H., brachial plexus injury received November, 1919, explored January, 1920—all roots intact, but three were thinned and part of one excised and end to end suture done (left arm). Massage and electricity given throughout course.

TABLE 5.—FINDINGS IN CASE 2

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Anesthesia to Pin Prick	Remarks
4/23/20	Biceps brachii	0.00033	75	Fair	None	Up to just above elbow posterior and about 6 cm. anterior; sensation of current and muscle contractions present down to elbow	Because of progressive shortening of current duration as arm was ascended a good prognosis given; no reaction to faradism below shoulder
	Teres major	0.00033	75	Fair	None		
	Median nerve	0.00065	75	Fair			
	Ulnar nerve	0.00065	75	Fair			
	Flexor carpi radialis	0.009	75	Good	None		
	Extensor indicis proprius and muscles of hand	0.009	75	Fair	None		
5/13/20	Biceps	0.00033	72	As before	As before	As before	Faradism negative in all
	Ulnar nerve	0.00065	75				
	Median nerve	0.00065	75				
	Flexor carpi radialis	0.009	75				
	Extensor indicis proprius and muscles of hand	0.009	75				
6/16/20	Biceps	0.00015	70	Poor	As before	As before	Faradism negative in all
	Median nerve	0.00033	75	Poor			
	Ulnar nerve	0.00033	75	Fair			
	Flexor carpi radialis	0.009	75	Good			
	Extensor indicis proprius and muscles of hand	0.009	75	Fair			
7/20/20	Biceps	0.00015	75	All stronger than on 6/16/20	Slight contractions of muscles above elbow	As before	Faradism positive in biceps, triceps and brachialis; negative in all others
	Triceps	0.00015	75				
	Brachialis	0.00015	75				
	Ulnar	0.00033	75				
	Flexor carpi radialis	0.009 and 0.0008 doubtful	75				
	Extensor indicis proprius and muscles of hand	0.009	75				
9/28/20	Biceps	0.00015	85	More active than on 7/20/20	Very slight flexion of elbow and supination; movements of shoulder girdle active	As before but pinching is felt numbly over entire lateral surface of forearm and hand but not on median surface	
	Triceps	0.00015	85				
	Brachialis	0.00015	85				
	Ulnar	0.00015	75				
	Flexor carpi radialis	0.00080	85				
	Extensor indicis proprius and extensors of wrist	0.00080	85				
	Flexor of wrist	0.00080	85				
	Muscles of hand	0.009	55				
11/30/20	Biceps, triceps, brachialis, ulnar	0.00015	85	Fair	Supination, pronation, flexion and extension of elbow abduction of arm weak but definite	As on 9/28/20 except anesthesia to just below elbow and pinching felt numbly over all of forearm and hand and first phalanx of fingers	
	Extensors of wrist	0.00080	92	Fair			
	Flexor carpi radialis and flexor of wrist	0.0008	92	Fair			
	Muscles of hand	0.009	92	Fair			
3/16/21	Biceps, triceps, brachialis and ulnar	0.00015	85	Good	As on 11/30 but much stronger; very slight flexion of fingers	From middle of hands distal; from elbow and midarm-medial down cannot distinguish between head and point of pin; deep muscle and joint sense corresponds in quality to above	Shoulder, elbow, wrist and finger joints show from 20-40% restriction of motion; atrophy of muscles of hand and forearm more marked since patient has not had treatment past 2 months; faradism as on 7/20/20
	Extensors of wrist	0.0008	100	Only fair			
	Flexor carpi radialis	0.0008	100	Only fair			
	Flexors of wrist	0.0008	100	Only fair			
	Muscles of hand	0.009	75	Only fair			

Summary: Shows a progressive lengthening of the minimum duration of current; therefore good prognosis given; a slow change of muscle complex to nerve-muscle complex in many muscles and a shortening of nerve-muscle complex in others; a return of voluntary movement in those muscles whose complex shortened to normal; a reduction in anesthesia; a gain with the chronomyometer of 3 months over faradism, 3 months over voluntary movements and 5 months over reduction of anesthesia.

CASE 3.—N. K., popliteal aneurysm ligated in upper part of popliteal space, June 6, 1920.

TABLE 6.—FINDINGS IN CASE 3

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Anesthesia to Pin Prick	Remarks
6/ 3/20	Right leg; all points	Infinite	70	None	None	Lower lateral surface of leg	No contractility or irritability of muscle—ischemid probably
	Right thigh and left leg	0.00033	50	Good	Normal	Decrease over balance	
6/ 7/20	Tibialis anticus	0.0008	70	Fair	None	That of sciatic nerve paralysis	Faradism negative
	All other on right leg	0.009	70	Fair	None		
6/15/20	All in right leg	Same on 6/7/20	...	.....	.....	.....	Faradism negative
8/13/20	All in right leg	0.0008	70	Fair	None	Same as 6/7/20 practically	Faradism negative
10/16/20	Right leg and anterior group	0.0006 definite 0.00033 doubtful	105	Good	Slight	Foot and ankle	Trophic ulcer on heel; patient has very acute purulent arthritis of right knee; faradism positive; some edema
	Posterior group (right leg)	0.00033	105	Good	Slight		
10/23/20	.....	.....	...	.....	.....	.....	Midthigh amputation right and nerve from knee to ankle studied histologically

Summary: Nerve-muscle complex returned 4 months ahead of faradism, between 2-4 months before sensory anesthesia decreased, and 2 months before voluntary contractions. On June 3, 1920, a diagnosis of ischemic paralysis was made but on June 7, 1920, it was clearly that of sciatic paralysis (incomplete). A good prognosis was given. The length of the nerve-muscle complex gradually shortened until 4 months later when it was within normal limits for the muscles of the leg and slight voluntary contractions were present; those of foot not tested. Histologically, cross sections of tibial nerve showed approximately normal number of axis cylinders 5 cm. below knee, fewer 10 cm. below knee and so on until at ankle there was about one to a low power field. This checks up with the electrical findings.

CASE 4.—G. B., fracture of distal end of left femur on March 21, 1920.

TABLE 7.—FINDINGS IN CASE 4

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Anesthesia to Pin Prick	Remarks
6/23/20	All left leg	0.009	70	Fair	None in foot	That of a sciatic lesion	Impression-degeneration of sciatic nerve below popliteal space; massage and electricity and wait 2 months
	All left thigh	0.00033	70	Very active	Normal	None	
7/26/20	All of left leg	0.009	105	Weak	None	Same	No massage or electricity for 2 weeks, therefore much edema; faradism negative
8/26/20	Left tibialis anticus, peroneus	{0.0008 sure {0.0006 doubtful	75	Weak	None	Smaller but of same shape as on 6/23	Prognosis given, good and no operation advised; faradism negative
	Gastrocnemius and soleus	0.0008	75	Weak	None		
9/18/20	All of left leg	{0.00033 {0.009	100 40	Fair	Very slight if any	Sole of foot and about 1½ inches up on foot all around	Few trophic ulcers; faradism negative
11/17/20	All of left leg	0.00015	90	Fair	Contraction of muscles but ankylosis prevents movements	Over all of toes and distal half of sole	Faradism negative (cyanosis of leg below knee after baking); patient could not stand strong enough faradism to cause contractions
2/ 2/21	All of left leg	0.00015	90	Good	Good contractions	Over all of toes with blisters and cyanosis	Faradism positive

Summary: Muscle complex changed to the longest nerve-muscle complex in 2 months after first examination and this shortened to normal in 3 months; area of anesthesia became smaller parallel to decrease in duration of current but both showed regeneration from 1 to 2 months before voluntary contractions. When patient was first seen a complete sciatic paralysis was diagnosed, but the prognosis was withheld for 2 months (5 months after injury) to allow regenerating fibers (if present) to reach the muscles and skin of the leg. At the end of this time the anesthesia had decreased and duration of current showed a long nerve-muscle complex; therefore a good prognosis was given without an exploratory operation. This prognosis was followed by loss of anesthesia, normal nerve-muscle complex and voluntary contractions 3 months later at which time the patient could not stand a strong enough faradic current to cause contractions (gain over faradism of at least 3 months, over voluntary movements at least 23 days). On September 18 response to 0.00033 second at 100 V and 0.009 second at 40 V. 40 V = 2 × rheobasic voltage.

CASE 5.—P. G., poliomyelitis of one and one half years' duration; baking and massage past six months; right leg, poor flexion of foot, slight extension of big toe, poor extension of thigh, inversion and eversion of foot gone, flexion of knee gone.

TABLE 8.—FINDINGS IN CASE 5

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contractions	Voluntary Contractions
6/26/20	Gluteus maximus	0.00065	95	Fair	?
	Biceps femoris long head	0.009	95	Fair	No
	Semitendinous	0.009	95	Fair	No
	Semimembranous	0.009	95	Fair	No
	Gastrocnemius	0.0008	95	Fair	No
	Soleus	0.0008	95	Fair	No
	Vastus lateralis	0.00015-33	95	Fair	Yes
	Vastus medialis	0.00015-33	95	Fair	Yes
	Rectus femoris	0.00015-33	95	Fair	Yes
	Tibialis anticus	0.009	95	Fair	No
	Extensor hallicus longus	0.009	95	Fair	No
	Peronei	0.0008	95	Fair	Slight
8/ 3/20	Gluteus maximus	0.00015	95	Fair	Slight
	Biceps femoris	0.00015	95	Fair	Slight
	Semimembranous	0.00015	95	Fair	Slight
	Semitendenous	0.00015	95	Fair	Slight
	All others as on 6/26				

Summary: The muscle and slow nerve-muscle complexes explain the losses or impaired function of various muscles; by referring back to the origin of the nerves supplying the affected muscles the lesion is in L 4 and 5, S 1, 2 and 3 segments of the cord; muscles of thigh changed from muscle to rapid nerve-muscle complexes as slight voluntary movements returned.

CASE 6.—F., Bell's palsy left—onset July 30.

TABLE 9.—FINDINGS IN CASE 6

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Remarks
8/ 2/20	All of right and left facial	0.00015	50	Good	None on left	Faradism present
8/ 4/20	All of left facial	0.00015	50	Good	None on left	Faradism present
8/ 9/20	All of left facial	0.00015	50	Good	None on left	Faradism present
8/25/20	All of left facial	0.00015	50	Good	Slight	Faradism present
10/15/20	All of left facial	0.00015	50	Good	Good	Faradism present

Summary: Paralysis came on over night, nerve-muscle response to both chronomyometer and faradic tests at all times but voluntary movement did not return for over three weeks. Good prognosis given on first examination. Voluntary contractions returned slightly in three weeks, definitely in six weeks.

CASE 7.—M. W., left facial paralysis, complete, following suppurative parotiditis and open drainage Nov. 4, 1920. Paralysis first noted December 7.

CASE 8.—R. T., left facial paralysis, onset July 2, 1920.

CASE 9.—H. R., right facial paralysis; onset sudden, July 15, 1920; examined July 24, 1920; slight voluntary power in orbicularis oculi only; nerve-muscle response 0.00015 second with good contractions over entire right and left face; given good prognosis for early recovery possibly within two weeks. Aug. 3, 1920,

(ten days later) a letter from the patient stated that he had the normal use of all muscles of the face. He was not tested with faradism which test no doubt would have been positive.

TABLE 10.—FINDINGS IN CASE 7

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Remarks
1/ 5/21	All of left facial:					
	Upper branch..	0.009	90	Weak	None	
	Mid branch.....	0.0008	90	Weak	None	
		0.009	30			
	Inferior branch.....	0.00015	30	Fair	Partial	
1/18/21	Upper branch.....	0.009	30	Weak	None	Patient massaging own face
	Mid branch.....	0.0008	90	Weak	None	
	Inferior branch.....	0.00015	30	Fair	Partial	
2/ 2/21	Upper branch.....	.....	..	.....	None	Patient massaging own face
	Mid branch.....	.....	..	.....	Slight	
	Inferior branch.....	.....	..	.....	Fair	
2/28/21	Upper branch.....	0.0008	80	Weak	None	Patient massaging own face
	Mid branch.....	0.00033	80	Weak	Fair	
	Inferior branch.....	0.00015	30	Weak	Good	
4/15/21	Upper branch.....	0.00025	50	Weak	Slight	Patient massaging own face
	Mid branch.....	0.00011	30	Good	Fair	
	Inferior branch.....	0.00011	30	Good	Good	

Summary: Seen four weeks after paralysis noted; very slight voluntary control of muscles innervated by inferior branch. Good prognosis given on basis of good nerve-muscle complex in inferior branch and slow in mid branch, two weeks later some muscles in superior branch showed nerve muscle complex and two weeks after this the patient had some voluntary control of muscles innervated by lower two branches. Two months later there was rapid nerve muscle complex in all three branches and voluntary control in all three branches, though that in the superior branch was slight.

TABLE 11.—FINDINGS IN CASE 8

Dates of Examinations	Motor Points Tested	Duration of Current in Seconds	Approximate Strength of Current in Volts	Type of Contraction	Voluntary Contractions	Anesthesia to Pin-Prick	Remarks
7/23/20	All of left facial	0.009	20	Fair	Negative	None	Faradism negative
8/10/20	All of left facial	0.009	20	Fair	Negative	None	Faradism negative
8/28/20	All of left facial	0.0008	55	Fair	Negative	None	Faradism negative
9/20/20	All of left facial	0.00015	15	Fair	Very doubtful	None	Faradism positive; slight wrinkles left side of face
10/23/20	All of left facial	0.00015	15	Good	Present but weak	None	Faradism positive
11/15/20	.....	... ...	..	.....	Practically normal		

Summary: Patient seen three weeks after paralysis, etiology unknown but possibility of severed nerve trunk very remote; operation not warranted though no nerve-muscle complex present. Prognosis withheld until August 28, two months from onset, when beginning nerve-muscle complex developed, yet faradism negative. Faradism positive one month later when doubtful voluntary contractions were present and wrinkles of skin began to return. Gain over faradism one month, over voluntary contractions at least twenty-three days.

Summary: This case illustrates that normal nerve-muscle complex was present even when the muscles did not contract voluntarily and recovery was rapid.

CASE 10.—V. H., there was right facial paralysis in April, 1920, following a mastoid operation. Examination was made Oct. 21, 1920; paresis of middle and inferior branches and complete paralysis of superior branch; nerve-muscle

complex of inferior and middle branches, 0.00015 second, superior branch 0.009 second with 50 volts and 0.008 with 70 volts; faradism positive in inferior and middle branches, but negative in the superior. Jan. 29, 1921, the patient's letter stated that he had good voluntary movement over the middle and inferior branches, but slight in superior branches.

Summary: Because of beginning nerve-muscle complex with 70 volts, we felt entire nerve would recover, and the later report seems to indicate that it is occurring.

Case 11.—H., partial left facial paralysis following brain abscess of left temporal lobe on Dec. 1, 1920. Paralysis noted Jan. 8, 1921. Examined Jan. 13, 1921. Only slight voluntary control of upper branch; no voluntary control of the two lower branches; good reaction in all the branches with 0.00015 at 10 volts; Jan. 8, 1921, good voluntary control of all branches.

Summary: Rapid nerve-muscle complex was present at height of paralysis, so that good prognosis was given and complete recovery followed in a week.

Case 12.—G. B., general polyneuritis (?), examined Nov. 20, 1920; all voluntary movements of left leg and ankle gone and only slight movement of third toe. Anterior and posterior groups of muscles of the left leg reacted only at 0.009 second with 80 volts. Right leg, movements of ankle and toes were slightly present and muscles reacted to 0.00033 second with 95 volts. No contractions elicited in either leg with faradic or galvanic current because the hyperalgesia made it too painful to use a strong enough current to bring out a reaction with either current. Burning sensation which patient could stand comfortably was present with the chronomyometer tests.

Summary: The chronomyometer reactions corresponded to the paralysis or paresis present. This case illustrates that electrical tests may be made on a patient with the chronomyometer when it is not possible to test the patient by ordinary methods because they are too painful.

Case 13.—E. K., median and ulnar secondary suture; extensor digitorum communis, 0.00015 second and good voluntary power; extensor carpi ulnaris 0.00033 second and poor voluntary power; other muscles of the forearm were normal. Ten months later, 0.00015 second in all muscles of the forearm and hand at 25 volts except lumbricales, which required 95 volts and good voluntary power in all muscles except the lumbricales.

Summary: This case illustrates the increase in voluntary power paralleled by shortening of the nerve-muscle complex.

Case 14.—A. W. G., gunshot wound through the lumbar region. Current strength 65 volts, right leg: obturator nerve, 0.00033 second; femoral nerve, 0.00015 second; biceps muscle, 0.00033 second; tibial nerve, 0.00033 second; peroneal nerve, no response; tibialis anticus muscle, 0.009 second; peronei, 0.009 second; soleus, 0.00015 second; gastrocnemius, 0.00015 second. Left leg: sciatic nerve, 0.00015 second; peroneal nerve, negative; tibial nerve, 0.00015 second; biceps femoris, 0.00015 second; tibialis anticus, 0.0008 second; peronei, 0.0008 second.

Summary: Examination revealed a bilateral injury to the sacral plexus affecting the roots which supply primarily the peroneal nerves, especially on the right side.

Case 15.—A. G., median and partial ulnar in axilla; repaired two years ago. Examined June 30, 1920. All motor points 0.00015 second with 70 volts and limited function, but sensory regeneration practically normal; on Feb. 20, 1921, practically normal function.

Summary: Rapid nerve-muscle complex indicated that problem of regaining function was one of reeducation which was confirmed by observation eight months later.

CASE 16.—D. L. R., ulnar injury above the wrist six years ago; repaired March, 1920; tested July 9, 1920; some sensations in little finger. There was marked atrophy of muscles of hand, therefore electrical testing was difficult. Reaction to 0.0008 second and doubtful voluntary movements of ulnar muscles.

CASE 17.—M. R., examined July 20, 1920; reduced congenital dislocated hip with sciatic nerve injury left; reduced three years ago, in cast three months, anesthesia from knee down, weak movements of knee and paralysis of foot when cast was removed. Reaction of gluteals, 0.00015 second; upper thigh, 0.00033; lower thigh, 0.00065; below the knee, 0.0008 second posteriorly, and 0.009 anteriorly. Right leg showed normal reaction. March 19, 1921: All of left thigh, 0.00015 second, soleus and gastrocnemius, 0.0008 second, tibialis anticus and peroneii, 0.009 second with poor reaction, plantaris longus, 0.009 second. Anesthesia to pin prick below ankle, but deep sensation present down to tips of toes. Voluntary Movements: Thigh good, knee fair, ankle only slight extension, foot inverted with toe drop.

Summary: In eight months nerve-muscle complex of thigh shortened, and parellel with this the voluntary power increased; no signs of improvement below the knee except a decrease in the anesthesia and a slight extension of the ankle which shows that the slow nerve-muscle complex means some innervation.

CASE 18.—S., spastic paraplegia from a spinal cord cyst. Reaction of all motor points of both legs, 0.00015 second with 20 volts.

Summary: This case shows that the paralysis due to injury of the upper neuron did not alter the electrical reactions of the lower neuron.

CASE 19.—J. B., back injured July 20, 1920. Tested Sept. 10, 1920. Paraplegia from the level of iliac spines down, with anesthesia to touch, and only the muscle complex 0.009 with 100 volts.

Summary: Complete degeneration of the lower cord and peripheral nerves.

CASE 20.—R. K., amyotonia congenitalis (?). No nerve-muscle or muscle response in either leg except in the rectus femoris right (0.00015 second, good response); sartorius right (0.00015 second, fair response) vastus externus, right (0.0008 second, weak response). Voluntary movements only in rectus femoris and sartorius. No anesthesia or trophic changes. Legs about half normal size, but had appearance of having normal sized muscle for that sized leg.

Summary: The electrical response was parellel to voluntary movements and showed a degeneration of the motor nerves and their muscles.

CASE 21.—M., lower spinal injury followed immediately by operation; complete paralysis of both legs since accident one and one-half years ago; marked atrophy of muscles of both legs; no voluntary movements; trophic ulcers and limitation of motion of joints of both legs, no muscle or nerve-muscle response in either leg; anesthesia up to mid-thigh. The patient showed complete loss of irritable nerves or muscles eighteen months after injury.

DISCUSSION

Previous workers who have studied regeneration of nerves by observations on the chronaxie have hoped to find a method by which at a single examination it could be determined that the process of regeneration was going on. In consequence Adrian [14] has arrived at the

conclusion that though chronaxie is of great physiologic interest, it has no practical value. Thus he says "The presence or absence of excitable nerve fibers can be detected well enough by the ordinary faradic coil and except for purposes of research there is little to be gained by the use of condensers or any other more elaborate method." In Lapicque's [13] published papers he has observed that chronaxie lengthens as degeneration progresses, but we find no statement indicating that he ever studied chronaxie changes in regenerating nerves. Furthermore, in all his studies he seems to disregard the variation in the strength of the current necessary to bring out chronaxie.

In the work recorded in this paper repeated observations were made with the chronomyometer during the regeneration and degeneration of nerves. Thus it has been possible to show that the nerve-muscle complex becomes shorter during the period of regeneration and that it lengthens during the degeneration of a nerve.

The only instance of repeated observations of chronaxie that we have been able to find any record of is a single case of Adrian's [14] on a facial paralysis in which he made observations on the orbicularis oculi and observed a gradual shortening of the chronaxie from 0.0075 second to 0.0003 second. In other words, the chronaxie shortened about twenty times, yet Adrian does not seem to consider this observation at all significant. Possibly this was due to the fact that voluntary movement was present when the chronaxie was 0.0075 second. In our own observations we have shown that far more important than chronaxie are the changes in the minimum duration of the current in so far as it brings out the progressive shortening of the nerve-muscle complex that occurs when a nerve regenerates.

It is, of course, apparent that until a regenerating nerve has reached a muscle one cannot hope to detect any changes in the minimum duration of the current. Thus the time between the appearance of the muscle complex following a nerve injury and the reappearance of the nerve-muscle complex will vary with the distance between the site of the nerve injury and the nearest muscle to be innervated by the nerve under consideration. When a nerve injury case is seen early (after about three weeks) it is of great value to know whether only the muscle complex is present, for this would indicate that the nerve has completely degenerated; following this, however, no observations on minimum duration are of value until the nerve has reached the nearest muscle. When a nerve, therefore, has a great distance to grow, which would mean months before one could hope to get any evidence of beginning regeneration, an early exploration seems desirable, but if the nerve has only a short distance to grow to the nearest muscle, exploration might well be deferred until the average time for regeneration has

elapsed (about 1 cm. a week). Where the principal disability is due to atrophy of small muscles, as in an ulnar or musculospiral paralysis, it may be unwise to defer operation.

If at exploration the nature of the lesion is still uncertain, another method suggested by one of us (J. Y. M.) of determining whether the axis cylinders have grown through the lesion may be employed. This subject was discussed in another paper which appeared in the November issue of *Archives of Surgery*.

SUMMARY AND CONCLUSIONS

The following points seem to us to be the most important ones derived from this study:

1. The chronomyometer is a practical instrument for detecting regeneration of nerves in clinical cases.

2. The determination of the minimum duration of a current necessary to stimulate a muscle is of greater value than determining the chronaxie.

3. The nerve-muscle complex after a nerve has been cut has always disappeared in animals within seventy-six hours and in one human case by the fifth day.

4. As regeneration occurs the nerve-muscle complex shortens gradually. This has been demonstrated both in animal experiments and clinical cases.

5. The nerve-muscle complex begins to appear from one to six months before faradic response, from one week to three months before voluntary contractions and from zero to five months before contraction of the area of anesthesia.

6. The variation in the minimum duration of the current necessary to stimulate muscles along the distribution of a nerve as well as repeated tests of one muscle may be of value in determining the regeneration of a nerve.

7. The faradic response of muscles and voluntary movements do not return until the nerve-muscle complex has reached normal limits.

8. Chronomyometer tests are of value as soon as a nerve has degenerated (five days or less) and as soon as regenerating fibers have reached the nearest muscle. This time can be estimated with fair accuracy by measuring the distance between the lesion and the nearest motor point.

9. The method herein described offers a more accurate means of determining much earlier the progress of nerve regeneration. Combined with a study of sensory changes, trophic disturbances, etc., it is a distinct aid in the prognosis and treatment of nerve injuries.

DISCUSSION

Dr. Sanger Brown of Kenilworth said that in the early eighties of the last century it was rather enthusiastically taught in some quarters that electricity might be used in paralysis due to a nerve lesion to hasten recovery, and no doubt at the present time it is quite extensively used for this purpose. Since these reported investigations seem to have been carried out with such exceptional thoroughness, he would like to ask whether the writers of the paper had any suggestions to offer on this point. He also asked whether the methods employed in testing would have any injurious effect on nerve lesion at any stage, because no matter how absurd such a question might appear, it was likely to be raised in medicolegal cases, where such tests were used.

Dr. M. Allen Starr of New York thought that the value of Dr. Sachs' paper could not be emphasized too greatly, because it had a medicolegal application. He said that occasionally he was asked to decide cases before the New York State Commission for Compensation for Injuries, and he recalled a case seen recently in which this would have decided definitely whether the man was going to recover from a serious brachial palsy or whether he was permanently disabled.

Dr. Isador Abrahamson, New York, asked whether Dr. Sachs, with his newer method of examination had studied its relationship to the incidence of such sequelae of facial palsy as contracture and hemispasms.

Dr. Ernest Sachs of St. Louis, in closing, said regarding the therapeutic effect, that this method had been of distinct value. A woman, who had a gash across her face, with complete facial paralysis, went to see him. The question was whether or not the facial nerves had been cut. Heretofore the only method of determining this was to wait and see whether the nerves would recover. By testing with this method, however, they found that the long nerve muscle complex was present. She was given a good prognosis, and in four months began to move her face.

They have tested now about thirty-five clinical cases of all sorts, and they have found that when the first evidence of the nerve muscle complex appearing is obtained, a complete regeneration of the nerve is certain.

Regarding Dr. Abrahamson's question about facial spasm, Dr. Sachs said they had had no experience with this condition.

THERAPY IN NEUROSYPHILIS, WITH PARTICULAR REFERENCE TO INTRASPINAL THERAPY *

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The pressing need of more effective therapy in neurosyphilis has stimulated syphilographers and neurologists to elaborate methods expressly adapted to combat the disease. These methods may be roughly divided into: (1) intensive antisyphilitic medication, including arsphenamin given intravenously, mercury and the iodids; (2) drainage of the spinal fluid, following a previous intravenous injection of arsphenamin; (3) the intraspinal methods of Swift and Ellis, Ogilvie and Byrnes. Each of these methods has produced good clinical results in the hands of competent observers, but each has certain inherent defects. Sachs [1] and his co-workers feel strongly that intravenous treatments will clear up such cases as are amenable to medication. Dercum [2] has obtained favorable results from complete drainage of the spinal fluid. Swift,[3] Ogilvie, Byrnes and Fordyce report good results following intraspinal procedures. Other than an inclination to advise intraspinal medication in difficult cases, there has been little tendency in the literature on the subject to indicate the type of therapy most likely to benefit a particular case.

That such individualizing, if it could be accomplished, would be of marked benefit is quite obvious. The intensive methods are the simpler, they require no specialized technic and therefore can be used more generally by the profession. They are less distressing and time-consuming to the patient.

Drainage is next in point of simplicity and presents no drawback, except lumbar puncture headaches and the inconvenience of the punc-

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, June, 1921, by Dr. H. G. Mehrtens.

1. Sachs, B.: The Truths About Intraspinal Treatment. J. A. M. A. **69**:681 (Sept. 1) 1917.

2. Dercum, F. X.: Functions of Cerebrospinal Fluid with Special Consideration of Spinal Drainage and of Intraspinal Injections of Arsphenamized Serum, Arch. Neurol. & Psychiat. **3**:230 (March) 1920.

3. Swift, Homer F.: Intraspinal Treatment of Syphilis of the Central Nervous System, J. A. M. A. **69**:2092 (Dec. 22) 1917.

ture. The intraspinal methods require a rigid technic, more time and apparatus and are more distressing to the patient. The patient's interests are best subserved when these methods are applied only when simpler methods are unavailing.

PATHOLOGIC LESIONS FROM THE STANDPOINT OF THERAPY

We may now consider the various pathologic lesions, grouped under the caption of neurosyphilis, from the standpoint of therapy. For our purposes the classification of Southard and Solomon [4] is especially useful. They note these types: meningeal, vascular, parenchymatous, meningovascular and diffuse (meningo-vasculo-parenchymatous). Of these divisions, the vascular and meningovascular types would seem to be best treated by way of the blood stream.

The therapy of epidemic cerebrospinal meningitis demonstrated that certain meningeal lesions are most effectively combated by allowing the therapeutic agent direct access to the lesion by way of the subarachnoid space.

It seems improbable, even on theoretic grounds, that intraspinal treatment is indicated in every case of meningeal syphilis, for it has been repeatedly shown that arsenic injected intravenously appears in the spinal fluid in from 25 to 40 per cent. of the cases treated. Whether or not one is inclined to place much emphasis on the importance of the presence of arsenic in the spinal fluid, it would seem at least to indicate good permeability in the minority of cases. These patients should do well on intensive therapy alone.

Complete spinal fluid drainage has established itself as a valuable therapeutic measure in tuberculous as well as epidemic meningitis. Dercum believes that the good results obtained in neurosyphilis may be explained on a basis of a reduction of increased intradural pressure, a lavage of the dural spaces, and a relative vascularity of the meninges and cord with consequent improved nutrition.

EFFECT OF INTRASPINAL THERAPY ON MENINGES

Flexner and Amoss,[5] in their work on poliomyelitis, demonstrated a let-down in the barriers between the blood and spinal fluid. This let-down could be produced by a variety of irritants, ranging from the intraspinal injection of blood serum to lumbar puncture. They felt that the permeability of the meninges was dependent on the irritation.

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4. Southard and Solomon: Neurosyphilis, Case History Series, Boston, W. M. Leonard.

5. Flexner, S. and Amoss, H. L.: Neutralizing Substances in Poliomyelitis, J. Exper. Med. **25**:499 (April) 1917.

Continuing along these lines, one[6] of us undertook some experiments to determine the natural permeability of the meninges to antisyphilitic medication. It was found that arsenic could be detected in the spinal fluid in 42 per cent. of patients receiving intravenous injections of arsphenamin. This was a slightly higher percentage than was found by Solomon and Rieger.[7]

The meninges were then irritated by the patient's own serum, as must normally occur when an intraspinal injection is given. This irritation showed itself in a spinal fluid pleocytosis of from 100 to 2,300 cells per cubic millimeter. The arsenic estimated was found in exceedingly small amounts, averaging 0.036 mg. per cubic centimeter in the unirritated cases and 0.1 mg. per cubic centimeter in the irritated ones. It has been objected that the amount of arsenic found in either case was too small for accurate estimation, and might well be within the limits of error of the chemical methods employed; therefore, at the present time we are still unable to state definitely that Flexner's ideas concerning the therapeutic value of irritation can be applied clinically to man.

We can feel certain that intraspinal therapy produces meningeal irritation of a most intense character, that the hyperemia of the meninges and exudation of the serum following this irritation is more intense than that obtained by any other means commonly employed.

CLINICAL RESULTS

Turning now to the clinical results, let us determine when possible, how far these theoretic considerations are supported by the facts observed here. Our material consists of 208 cases of neurosyphilis treated by intraspinal medication since 1915. Seventy-five patients were given thorough treatment, receiving from six to eighteen intraspinal injections. During that time 1,500 intraspinal injections were given. This material is unusual in that, previous to that time, all neurosyphilitic patients in the Stanford Clinics were treated by the intravenous and intramuscular methods. We therefore had the opportunity of estimating the results from this form of therapy without the addition of other procedures.

Ignorance of the language, inability to understand the importance of continuing treatment, combined with financial difficulties, resulted in many incompletely treated cases in our clinical material.

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6. Mehrtens, H. G., and MacArthur, C. G.: Therapy of Neurosyphilis Judged by Arsenic Penetration of the Meninges, Arch. Neurol. & Psychiat. **2**:369 (Oct.) 1919.

7. Solomon, H. C., and Rieger, J. B.: Circulation of Arsenic in the Cerebrospinal Fluid, J. A. M. A. **71**:15 (July 6) 1918.

INTRAVENOUS AND INTRAMUSCULAR THERAPY

In considering the results in patients treated with intravenous and intramuscular therapy, it must be noted that a few of the treatments extended over the whole year and, therefore, the patients did not receive treatment in quite so intensive a form as patients with intraspinal cases.

Some clinical improvement was obtained in the majority of cases. Serologically only 19 per cent. cleared up entirely, that is, the Wassermann reaction was negative in all dilutions, the globulin normal and the cell count three or less. Of these, one patient became positive again serologically in a few months. The vascular and meningovascular lesions were more amenable to therapy, as judged by both symptomatic and serologic improvement. The parenchymatous lesions showed no improvement either clinically or serologically.

TABLE 1.—CASES TREATED WITH INTRAVENOUS AND INTRAMUSCULAR THERAPY

		Clinical Results				Serologic Results					
		Improved		Not Improved		Clear		Improved		Not Improved	
Diagnosis	Number of Cases	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
Tabes	14	12	85	2	15	2	14	10	71	2	14
Cerebrospinal syphilis	11	11	100	0	..	3	27	8	73	0	
Paresis	1	0	...	1	..	0	..	0	..	1	..
Totals	26	23	88	3	12	5	19	18	69	3	12

DRAINAGE THERAPY

Intravenous medication plus drainage was used in thirty-six cases (from four to ten drainages). Unfortunately this form of therapy was combined with intraspinal medication in most cases, making conclusions unreliable. The change to the Swift-Ellis and Byrnes methods was frequently made at the request of the patients, who, as a rule, seemed to prefer the intraspinal methods, or improvement seemed so slow that the staff felt that the patient should receive the most vigorous treatment possible. Our impressions of the results thus obtained from drainage were: Symptoms dependent on spinal fluid pressure, such as headaches and other complaints which could be traced to intercranial pressure, were ameliorated. The serologic improvement was considerably better than that which followed simple intravenous medication. In brief, we felt that drainage is definitely more effective than intravenous or intramuscular therapy alone. There were cases, however, which seemed resistant to drainage but which yielded to intraspinal therapy.

RESULTS OF INTRASPINAL THERAPY

The intraspinal therapy used consisted of the Swift-Ellis, Byrnes and Ogilvie methods and the modifications suggested by one [6] of us. Table 2 shows that every case of cerebrospinal syphilis improved symptomatically; 75 per cent. became negative serologically. The tabetic patients, as a whole, did less well, but symptomatically 55 per cent. improved and more than 40 per cent. became clear serologically.

Patients with optic atrophies, generally far advanced before treatment began, showed no improvement. Patients with tabes without positive findings in the spinal fluid experienced little relief symptomatically. The lightning pains, crises and other symptoms continued as previously. If these two unfortunate types be omitted, 80 per cent. were improved symptomatically and 56 per cent. became clear serologically.

TABLE 2.—CASES TREATED WITH INTRASPINAL THERAPY

		Clinical Results				Serologic Results					
		Improved		Not Improved		Clear		Improved		Not Improved	
Diagnosis	Number of Cases	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.	Number	Per Cent.
Tabes	25	20	80	5	20	14	56	5	20	6	24
Optic atrophy	6	0	..	6	100	3	50	..	..	3	50
Arrested tabes	11	3	27	8	73	11	100				
Total tabes	42	23	55	19	45	28	66	5	11	9	21
Cerebrospinal syphilis	21	21	100	0	..	16	75	2	9	3	14
Paresis	12	4	33	8	67	3	25	3	25	6	50
Total cases	75	48	64	27	36	47	63	10	13	18	24

Five patients were given intraspinal treatment when ten or more intravenous injections of arsphenamin plus intramuscular mercury salicylate had previously failed to render the spinal fluid normal. Of these, three tabetic patients became entirely clear after four, six and seven further intraspinal injections, and remained clear. One of these cases had previously been drained five times in addition to the intraspinal treatment, and still showed a positive Wassermann and a pleocytosis. One case (tabetic) with optic atrophy showed a negative spinal fluid after six additional intraspinal injections. No clinical improvement was obtained. One patient with taboparesis showed no clinical or serologic improvement after eight additional intraspinal treatments. The results obtained in this rather small group would suggest that intraspinal therapy is the most potent means we now have of securing a normal spinal fluid. It also has its limitations.

Patients with paresis, as a whole, did poorly. Three cases so diagnosed clinically and serologically, cleared up entirely; the patients went back to work and have remained serologically clear for four, nine and twelve months, respectively. Possibly only a remission was

secured, but the fact remains that improvement did occur after treatment. At any rate, these cases emphasize the importance of treatment when there is any doubt about the diagnosis.

Disadvantages of Intraspinal Treatment.—The disadvantages of intraspinal methods may be divided into the discomforts incident to the method, pain, fever, occasionally nausea and difficulty in passing urine for twenty-four hours. That none of these features are of serious importance to the patient is demonstrated by the efforts of the patients to continue treatment in spite of many physical and financial handicaps. Patients requested intraspinal treatments who previously had been treated intravenously or by drainage.

The complications in 1,500 treatments were:

In one case in which the Byrnes treatment was used, the intern ran the mercuric chlorid up to $\frac{1}{25}$ of a grain. (Our custom is never to use more than $\frac{1}{50}$ of a grain.) The result was bladder incontinence which has not improved in two years. One patient with paresis died suddenly of apoplexy following preliminary intravenous injection of arsphenamin. One patient with tabes died immediately following the intravenous injection of arsphenamin given before the intraspinal treatment. Two tabetic patients previously treated with six and eight intraspinal injections, respectively, still had positive findings in the spinal fluid. They were both given the usual preliminary arsphenamin injection intravenously. Either because of the peculiarity of the drug or the way it was mixed both patients developed respiratory distress and pulmonary edema, followed in three and four days, respectively, by chest signs of pneumonia and fever. After recovery from these complications, intraspinal therapy was resumed. One patient was found to possess a normal spinal fluid, the other case became normal following one more intraspinal injection.

These complications emphasized the fact previously brought out, particularly in relation to paresis, that intercurrent infections tend to influence favorably the course of neurosyphilis. It even seemed possible that the fever, which almost invariably occurred following intraspinal injections, might itself be responsible for some of the good therapeutic results.

Thus our experience with 1,500 treatments has been as free from complications as a corresponding number of intravenous injections of arsphenamin alone.

During the course of intraspinal treatments it occasionally became impossible to give intravenous injections because of thrombosis of the veins. In order to get the arsphenamin into the circulation in these cases, one [8] of us used massive doses of neo-arsphenamin (4 gm.)

8. Mehrtens, Henry G.: Rectal Injection of Massive Doses of Neo-Arsphenamin, J. A. M. A. **76**:574 (Feb. 26) 1921.

injected rectally. In over 300 rectal injections there were no disagreeable sequelae, except in one patient who had developed a very severe case of exfoliative dermatitis. This condition had previously occurred in a milder form in the same patient, following an intravenous injection.

Following these massive rectal injections of neo-arsphenamin, arsenic was found in the spinal fluid in about the same concentration as after an injection of 0.6 gm. of arsphenamin injected intravenously.

In sixteen cases we were forced to substitute the rectal for the intravenous injection of arsphenamin in intraspinal treatments. The results are not ready for final evaluation but are sufficiently encouraging to warrant the use of rectal injection of arsphenamin when the intravenous route is impracticable.

CONCLUSIONS

Intravenous and intramuscular therapy caused symptomatic improvement in the majority of cases. Serologically only 19 per cent. cleared up entirely. It was more efficacious in the meningeal, vasculomeningeal and diffuse types.

Drainage, in addition to the foregoing results, impressed us as a definite advance both in ability to ameliorate symptoms and in tendency to improve the spinal fluid pathology.

Intraspinal medication was superior to the intravenous and intramuscular methods in its effectiveness in clearing up the spinal fluid. Forty-eight per cent. of the cases became clear through the use of the intraspinal methods as compared to 19 per cent. following the intravenous method.

The most useful field for intraspinal therapy is that of the meningoparenchymatous types, including tabes. However, patients with optic atrophy and with tabes without meningeal reaction received no benefit. Patients with parenchymatous lesions (including paresis) did poorly, but 25 per cent. of the cases thus treated cleared up clinically and serologically. A remission, at least, was effected.

In our experience complications in intraspinal therapy are no more frequently met with than in intravenous medication.

Massive rectal injections of neo-arsphenamin (4 gm.) may be profitably substituted for arsphenamin given intravenously in intradural medication when intravenous injection is impracticable.

In the treatment of the individual case of neurosyphilis, it would therefore seem proper to begin with intensive intravenous and intramuscular medication, particularly in vascular, meningovascular and diffuse lesions. Failure to reduce spinal fluid findings to negative after a thorough trial should suggest the advisability of using more intensive methods. Drainage, combined with intravenous injections, again should be the procedure of choice when the facilities for more

complicated methods are lacking or when symptoms of increased spinal fluid pressure are distressing. The Swift-Ellis, Ogilvie or Byrnes method should be reserved for cases resistant to the foregoing efforts. These resistant cases will be found particularly in tabetic patients. Patients with optic atrophy and neurosyphilis without cerebrospinal fluid reaction receive no advantage from intraspinal medication. This was anticipated by one [9] of us in a pathologic study of such cases. Paretic patients should be given a trial. Patients with inadequate veins can profitably receive the arsenic in the form of massive rectal injections of neo-arsphenamin.

DISCUSSION

Dr. Walter F. Schaller of San Francisco thought that the interest in intraspinal therapy had been quite general. There had been a difference of opinion as to the efficacy of this method of treatment, indeed whether such treatment should be given at all. The objections, as far as he had been able to follow in the literature, had been a general expression of opinion rather than a comparison of groups of neurosyphilitic cases, treated intensively on the one hand by intravenous therapy and on the other by intraspinal therapy. The object of the paper was to present such a comparison for consideration. It was his opinion, based on this study, that the greatest field of usefulness of intraspinal therapy was in refractory cases which did not improve under the intensive intravenous and other specific medication, and especially in meningeal forms and early meningoparenchymatous forms of neurosyphilis. A number of patients treated intensively in the dermatologic clinic and developing nervous symptoms showed on cerebrospinal analysis positive fluids. These fluids were cleared serologically and the patients relieved symptomatically by intraspinal therapy. Conversely, patients with evident neurosyphilis with negative serology were not improved by intraspinal treatment. On theoretic consideration the latter result was anticipated a few years ago, which Dr. Schaller thought had been borne out by practice.

Dr. Israel Strauss of New York said that this paper interested him very much because he thought it was probably the first one written by a neurologist on this subject, after careful study and investigation. Most of the papers written on intraspinal therapy had been given by dermatologists and others who had not been competent to judge the results of the treatment because of their ignorance of the symptoms, and particularly of the course of neurosyphilis. This was the first paper which Dr. Strauss had ever heard on intraspinal medication, in which it was admitted that not merely was there discomfort but that there could be serious complications.

Furthermore, he was certain that the writers of this paper, although they did not quote the literature, knew there had been a number of cases in which death had followed the use of the method. Of course, this criticism applied to medicine in its general aspect because it is never known at what time a certain method or even a commonly used drug may find an individual who is susceptible and who suffers unusually from the therapeutic measure. Nevertheless, this treatment is fraught with considerable danger, even though

9. Schaller, W. F.: The Pathogenesis of Tabes Dorsalis, Arch. Neurol. & Psychiat. **1**:749 (June) 1919.

the writers of this paper, outside of the few instances they cited, were fortunate enough in not obtaining serious results.

Dr. Schaller stated that he limited the use of this method to the so-called meningeal type of syphilis, which statement he qualified by saying tabes. In certain cases of tabes, intravenous treatment did not give the desired relief from clinical symptoms, and in a few cases favorable results were obtained with the intradural method. It required a great deal of care in order to pick out the cases in which this method should be resorted to.

Dr. Harry W. Mitchell of Warren, Pa., said that for the past ten years about fifty cases of neurosyphilis, mostly of the paretic type, had been admitted annually to the Warren Hospital where the various methods of treatment had been adopted and the result checked by clinical and serologic criteria. He had noted during that period a larger percentage of remissions than was formerly seen in untreated paretic patients, but he was obliged to report that a large number of these patients showing remissions had ultimately been returned to the hospital where their disease had terminated fatally. As a result of his experience with this condition he regarded any improvement, either serologic or clinical, as temporary rather than permanent. This observation applied to cases so far advanced as to require commitment to a hospital for the insane. Possibly serologic determination of the condition before it had advanced to the point at which clinical diagnosis was positive might be followed by early, vigorous treatment resulting in actual "cure." This question, however, could be settled, in his opinion, only by prolonged treatment and observation of many such early cases. Pending such determination he could see no justification in speaking of improvement in paresis as anything more than a remission, and he believed that the word "cure" was no more applicable to these cases than it would be in the primary stage of syphilis following a short course of antisyphilitic treatment.

He felt possibly the best results that could be obtained from recent methods of diagnosis and treatment of neurosyphilis would be the recognition of serologic evidence of the disease in immediate relatives and starting treatment that might prevent the hopeless conditions.

He desired to know whether the readers of the paper had reason to present any different conclusions regarding neurosyphilis of the paretic type.

Dr. Walter F. Schaller said that he could speak definitely of the ultimate course of only three patients with paresis who showed remissions after intraspinal therapy. These patients were cleared serologically as well as arrested symptomatically and remained so as long as observed, in one case after a period of one year.

THE SIGNIFICANCE OF BIOLOGIC REACTIONS IN SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

WITH NOTES ON TREATMENT, ESPECIALLY INTRASPINAL *

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In a paper published in 1918, we [1] reported an intensive study of this subject in the neurologic wards and serologic laboratory of the Mount Sinai Hospital. We now desire to bring our experiences down to date and to express our present views. In the light of a tendency to overemphasize the importance of early spinal fluid reactions, we have deemed it expedient critically to review these reactions and to estimate their significance as regards changes in the nervous system, and their proper relation to the nature and intensity of treatment.

Our knowledge of cytodiagnosis of the cerebrospinal fluid began with the work of Widal, Ravaut and Sicard [2] in 1900. The addition of the Wassermann test, protein estimation and the colloidal gold test placed the diagnosis of syphilis of the central nervous system on a definite basis.

PRECLINICAL CHANGES IN SYPHILIS OF CENTRAL NERVOUS SYSTEM

Ravaut showed that during the primary and secondary stages of syphilis, there is in 68 per cent. of the cases, a latent nonsymptomatic meningitis, the only evidence of which is obtained by lumbar puncture. Dreyfus [3] found similar changes in almost 80 per cent. of the cases. These figures have been corroborated by numerous observers. Perhaps if puncture were performed often enough, such changes might be found in all early cases. The departure from the normal in the early stages consists principally of increase in the cell count, protein increase (albumin of globulin; phase 1 of Nonne), and occasionally, of the presence of the Wassermann reaction. These facts are incontrovertible; it is their interpretation and significance as indicating permanent or ephemeral change in the neuraxis that are in question.

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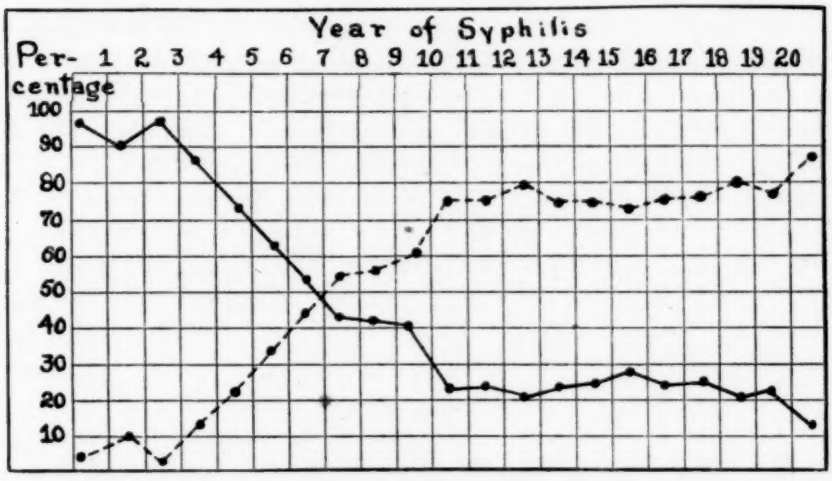
* Read before the Section on Nervous and Mental Diseases, at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

1. Kaliski, D. J., and Strauss, I.: On Syphilis of the Nervous System: Pathological, Serological and Clinical Criteria with Especial Reference to Treatment, Am. J. Syphilis **2**: No. 4, 1918.

2. Widal, F.; Sicard, A., and Ravaut, P.: Bull. Soc. méd. d. hôp de Paris, **18**:31-34, 1901; Ann. de dermat. et syph. **4**:1-14, 1903; Thèse de Paris, 1899.

3. Dreyfus, G. L.:Deutsch. med. Wchnschr. **45**:1293-1326, 1919; München med. Wchnschr. **66**:1374-1376, 1919.

According to Ravaut,[4] the early changes are transitory and of the nature of a septicemia, while those occurring later in the disease are definitely focal, usually of the nature of a meningovasculitis. These latent reactions are common up to the fourth year, as shown in the chart, diminishing from that time to the twelfth year, and remaining more or less stationary up to the twentieth year, when they again diminish. Persistence of the septicemic reaction after the third year is, in the opinion of Ravaut, evidence of involvement of the central nervous system, especially if all the reactions are positive (the number of cells, the globulin and Wassermann reaction). Thus, lumbar puncture



Relationship between biologic phenomena in spinal fluid and neurosyphilis (after Ravant).

Continuous line indicates presence of biologic changes in fluid and absence of clinical signs of neurosyphilis. Broken line indicates definite neurosyphilis accompanied by spinal fluid changes.

should be practiced during the first three years following infection, and if not then, at least at some period between the fourth and the tenth years, because then the ephemeral septicemic reactions tend to disappear while the clinical symptoms both objective and subjective tend to increase steadily. The importance of lumbar puncture is stressed by Ravaut as affording the only means of determining the presence of the latent meningitic condition, thus enabling one to take measures to eradicate it. To obtain evidence of the presence of syphilis in nervous or psychic disorders, especially if the blood reactions are negative, it is imperative.

Since we lack precise knowledge of the morphologic changes in early syphilis of the central nervous system, numerous hypothetic explanations have been offered for the presence in the spinal fluid of

4. Ravaut, P.: Presse méd. 1919, No. 57.

increased cells and protein and for the Wassermann reaction. According to Gennerich,[5] these changes are due to a histologic neurorecidive, and are found up to the third year in from 40 to 60 per cent. of early cases. Gennerich believes that the degree of change in the pia-arachnoid determines whether there will be eventual changes in the neuraxis of a metasyphilitic type, and he assumes that the pia-arachnoid serves as a barrier between the cerebrospinal fluid and the neuraxis. If the pia-arachnoid is pathologic, fluid diffusion occurs into the nervous tissue with the occurrence of pathologic changes.

Wechselmann,[6] in 1912, reported changes in the cerebrospinal fluid in the preroseolar period of syphilis. Plaut,[7] in considering the question of nervous involvement in early syphilis, speaks of the neurotropism of strains of spirochete, a belief engendered principally by the work of Fournier, Levaditi and Marie,[8] and concurred in by many workers, including Nichols and Hough,[9] and Reasoner.[10] Plaut describes the finding of spirochetes in the cerebrospinal fluid of patients with early syphilis, but is doubtful if this is the beginning of syphilis of the central nervous system. He says there is no proof that the presence of organisms in the fluid has any relation to late syphilis of the nervous system, since most of the cellular reactions disappear from the fluid spontaneously or as a result of early treatment. Both Gennerich and Mulzer have reported cases showing spirochetes in the fluid with negative biologic reactions. Wile and Hasley[11] believe that the so-called early preroseolar meningeal involvement which they found in forty-nine of two hundred and twenty-one cases "is for the most part transitory and without subsequent or permanent damage because in the majority the fluid was rapidly restored to normal under the influence of treatment and doubtless might have disappeared even without this treatment, like the early rash of syphilis." According to these authors, the appearance

5. Gennerich, W.: Berlin, in 1913; Hirschwald: Die Liquorveränderungen in den einzelnen Stadien der Syphilis, Deutsch. med. Wchnschr. **44**:1243, 1918.

6. Wechselmann, W.: Deutsch. med. Wchnschr. **38**:1446, 1912.

7. Plaut: Deutsch. med. Wchnschr. 1919, No. 48; Ztschr. f. d. ges. Neurol. u. Psychiat. **52**:193, 1919; Neurotherapy, **1**:76-88, 1919; **74**:988, 1920.

8. Levaditi, C., and Marie, A.: Rev. de méd. **37**:193, 1920.

9. Nichols, Henry J., and Hough, William H.: J. Exper. Med. **19**: No. 4, 1914; Demonstration of Spirocchaeta Pallida in the Cerebrospinal Fluid, J. A. M. A. **60**:108 (Jan. 11) 1913.

10. Reasoner, M. A.: Some Phases of Experimental Syphilis, J. A. M. A. **67**:1799-1805, 1916; Early Death from Cerebral Syphilis, with Successful Rabbit Inoculation, ibid, **66**:1917 (June 17) 1916.

11. Wile, U. J., and Hasley, C. K.: Involvement of Nervous System During Primary Stage of Syphilis, J. A. M. A. **76**:8 (Jan. 1) 1921; Serologic Cure (?) in the Light of Increasingly Sensitive Wassermann Tests, ibid. **72**:1526 (May 24) 1919.

of positive fluid findings before the Wassermann reaction in the blood becomes positive is evidence of generalization and not of neurotropism.

Stokes,[12] on the other hand, declares that early pleocytosis means involvement of the central nervous system in the sense of other tissue involvement in early syphilis. According to Dreyfus,[3] an observer of extensive experience, pathologic changes in the fluid indicate infection of the neuraxis. He believes that these can recede spontaneously only in the early stages. He found changes in 80 per cent. of his cases in the early stage, but includes as evidence of pathologic change, pressure above 180 mm. of mercury, to which we ascribe little or no importance. Fordyce [13] says the "activity of a syphilitic process in the central nervous system is indicated much earlier, more accurately and often only by the cytologic changes in the spinal fluid which should always supplement the physical examination."

Observers both here and abroad differ in the quantitative interpretation of spinal fluid findings. Thus, Cornaz [14] speaks of 2.4 cells per cubic millimeter as normal. Wile and Hasley [11] set an arbitrary normal of 8 cells and state that an increase of one or two cells can hardly be accepted as definite proof of involvement. Dreyfus [3] considers more than 5 cells as pathologic. Fildes, Parnell and Maitland [15] refuse to accept the high percentage of involvement published by Ravaut in view of the fact that abnormality is often based solely on the finding of 10 cells per cubic millimeter. They state that few of these cases show any abnormal signs such as would produce disability and many show no discoverable signs of disease.

Obviously, we need a standard of serologic normality, and this we have attempted to determine. We have examined thousands of fluids from patients with and without demonstrable involvement of the central nervous system from all types of specific and nonspecific disease, and we have come to look on from 8 to 10 lymphocytes per cubic millimeter as within the bounds of normality. These figures may appear too high, but we believe it safer to set a standard which, if exceeded, in all probability means meningitic change. Indeed, a slight increase above this normal in the absence of clinical signs or symptoms, unless accompanied by definitely increased globulin or by a positive Wassermann reaction, is probably of little significance. Persons without change in the central nervous system on whom spinal puncture is performed as a routine for general diagnosis, occasionally show as high as from 8 to 10 cells without globulin increase or other pathologic changes. On the other hand, definite syphilitic cases often show no increase in cells.

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12. Stokes, J. H.: M. Clinics N. America **2**: 1919.
13. Fordyce, J. A.: Am. J. M. Sc. March, 1921
14. Cornaz, Georges: J. Nerv. & Ment. Dis. **49**:282 (April) 1919.
15. Fildes, Parnell and Maitland: Brain **41**:255 (Nov.) 1918.

Cases of nonsyphilitic disease of the nervous system, such as multiple sclerosis, tumors of the cord and brain, chorea, epilepsies and other conditions frequently show 5 or more cells to the cubic millimeter without demonstrable meningitic change.[16] It is well to bear in mind that pleocytosis is often rapidly influenced by puncture only or antispecific treatment—a drop to normal limits from a fairly high count not being infrequent.

Increased protein content has always been looked on as evidence of organic change. It is supposed to be the earliest sign to appear and the last to disappear either spontaneously or as a result of treatment. On the whole, it may be said to go hand in hand with cell increase. It may be the only abnormal sign present in the fluid. By itself, globulin increase is of little significance in syphilis of the nervous system, and, unless looked on as a corroborative sign of other biologic phenomena, could be dispensed with in routine diagnosis.[17] A positive Wassermann reaction is of more significance, especially if the reaction is present in all dilutions from 0.1 c.c. upward. We have adopted 1 c.c. of fluid as the maximum amount. In the earliest stages of syphilis a positive reaction with large amounts of fluid is occasionally a transient phenomenon and may possibly be due to a transference of syphilitic reagin through the choroid plexus or other secreting surfaces of the pia-arachnoid. A persistently positive Wassermann reaction of the fluid, when present in early syphilis with pleocytosis and globulin increase, especially if the reaction is present in all dilutions, even in the absence of clinical symptoms referable to the central nervous system, should be looked on with suspicion, and calls for persistent treatment and lumbar puncture from time to time in the control of the therapy.

We consider the colloidal gold test of little value in early syphilis. In neurosyphilitic conditions it may serve to corroborate the other tests, but may be found in the absence of all other phenomena. As an indication of the eventual development of paretic changes, the so-called paretic curve should not be relied on. (We do not believe the paretic curve is diagnostic of general paresis, even though most paretic fluids give it.) In other words, given an early case of syphilis with serologic reactions persistently positive, even with a positive blood reaction and the so-called paretic curve, we believe that we cannot today prognosticate the development of parenchymatous syphilis.

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16. All counts should be made within an hour or two of puncture. A standard chamber should be used—the Fuchs-Rosenthal. If microscopic blood is present, the number of cells to an equivalent field should be noted. Frankly bloody fluids are not available for counts.

17. In estimating globulin increase it must be emphasized that only those fluids should be tested which are microscopically free from blood or contain at most a few red cells to the cubic millimeter.

In short, we believe that a mere increase in cells (pleocytosis) and in protein (globulin) with a transient Wassermann reaction point to irritation of the pia-arachnoid of the nature of a general septicemic reaction, temporary, and of doubtful significance as to the future involvement of the neuraxis.[18] The constant presence of cells, globulin and a definite Wassermann reaction in all dilutions in early syphilis indicate a more serious membrane involvement, possibly the earliest signs of a definite organic involvement of the neuraxis.[19] This view is tentative despite the fact that we have observed cases of syphilis from their incipiency with persistent serologic reactions with no subjective or objective clinical manifestations, over a period of ten years. The literature is curiously lacking in adequate statistical studies of cases with such persistent serologic reactions (serologic neurosyphilis) which have eventually developed definite involvement of the central nervous system.

It is a fact to remember that patients with vascular neurosyphilis may show no change in the cerebrospinal fluid—occasionally the blood Wassermann reaction is positive—but may suffer from serious damage due to vessel closure or rupture. These patients often are young people with early syphilis. They must be borne in mind when interpreting biologic data and when forming a prognosis based on tests of the cerebrospinal fluid.

INFECTION OF CENTRAL NERVOUS SYSTEM

One of the most important questions to be considered is the time of infection of the central nervous system. Morphologic studies are entirely lacking to decide this point. The biologic reactions are not conclusive. The idea that spirochetes enter the central nervous system in the early stages of the infection or not at all, and lie dormant in

18. If the early spinal fluid changes found in so high a percentage of syphilitic patients were indicative always or even frequently of invasion of the central nervous system by the spirochete, it is difficult to understand why symptomatic syphilis of the central nervous system is not more frequently encountered. We know that the neuraxis as a rule is susceptible to slight irritation or invasion by virulent organisms, and the nervous system in early syphilis should therefore in the circumstances respond with productive or other types of inflammation to an invader like the spirochete at a stage when the germ possesses its maximum virulence and the body resistance is at its lowest.

19. Fraser, A. R.: Am. J. Syphilis, **5**: No. 2, 1921. Fraser believes that the presence of serologic reactions in the fluid in the absence of clinical signs of involvement of the central nervous system points to the presence of an ample antibody supply with protection of the neuraxis, while the negative reaction means that the central nervous system has escaped involvement or that it has failed to react, which, he cannot tell. He believes that the positive early reactions in the fluid mean that the nervous system has reacted and therefore the prognosis is better.

relatively nonvascular regions but possess the potential power of later multiplication and assumption of virulence in response to some unknown stimulus, is still unproved. Is it not possible that spirochetes wander into the central nervous system from without and, finding a less resistant or more fertile soil, become active?[20] If spirochetes do lie latent in the neuraxis for years, as stated by Warthin,[21] what is responsible for this so-called immunity or failure to react? If immunity exists in syphilis, it has not been proved by the demonstration of immune bodies, antibodies, in the blood or spinal fluid. Clinically, soon after the chancre, even before the generalization of the virus, the body becomes insusceptible to reinoculation with syphilis and in the vast majority of cases remains so. Even in a patient with a case of syphilis of long standing when the "immunity" has become attenuated, if it ever does so, reinfection is rare, even after years of therapy and with absolutely negative serologic reactions, so that when we speak of immunity in syphilis, we are using an empiric term, not one scientifically exact.

We speak of antibodies produced in response to syphilitic infection being responsible for so-called immunity in syphilis, or of protective antibodies being present in the blood and spinal fluid, detectable by the Wassermann reaction. All this is hypothetical. There is no proof fulfilling immunologic requirements. We know that the antigen of the Wassermann reaction is of lipoidal nature and that the spirochete itself cannot thus be practically utilized; further, other protozoan diseases definitely not syphilitic give a high percentage of positive Wassermann reactions. Wassermann[22] himself has recently shown that there is present in the serum of syphilitic patients a substance that in the presence of complement enters into a reversible combination with the Wassermann antigen. The extract does not consist of the disease producing spirochetes or other protein material, but of alcohol-soluble, fat-like substances (lipoids) which are derived from animal organs.

20. Brown and Pearce showed that rabbits which recovered from clinical manifestations of syphilis are still infected and may continue to harbor virulent spirochetes in certain lymph nodes. The points of rest during latency are possibly chiefly the lymphoid tissues of the animal. Arzt and Kerl Wien. klin. Wchnschr. **27**:29 (July 16) 1914, reported finding in lesions about the genitals and in the regional lymphnodes of normal rabbits spirochetes absolutely identical morphologically with *Spirochaeta pallida*. These organisms were found in 26.9 per cent. of certain strains of normal rabbits. These observations have been confirmed by Jakobsthal, by Tomascewski, and in this country by Brown. These findings point out the necessity for great conservatism in accepting the conclusions drawn from rabbit inoculations and in our estimation call for a complete revision of all prior experimental inoculation work in rabbits.

21. Warthin, A. S. Am. J. M. Sc. **152**:508, 1916.

22. Recent experimental investigations on syphilis, Berlin letter, J. A. M. A. **76**:463, 1921.

This substance, which Wassermann recently isolated for the first time, is produced by the lipoid substance which must be present in large quantities in the blood of syphilitic patients. Wassermann attempts to prove a point heretofore unknown, namely: that lipoids as well as protein substances may produce genuine antibodies, thus explaining why the so-called antibodies present in syphilis were directed not against the specific spirochete of the disease, but against lipoids produced by spirochetal action on the tissue cells of the body. He asserts that the syphilitic patient thus suffers from an inversion of lipoid metabolism.[23]

In a recent paper Fraser [24] speaks of increase of neurosyphilis due to intensive treatment by arsphenamin in early syphilis after generalization causing rapid sterilization with loss of antibody protection for the central nervous system. He thinks that because of a lack of vigorous cell reaction, the spirochetes penetrate into relatively nonvascular areas of the central nervous system; that the central nervous system produces few or no antibodies and is dependent for protection on those produced as a result of the early general reaction. This is only a restatement of the views of Gennerich,[5] who holds that treatment, especially if insufficient, during the period of early dissemination, hinders generalization of the spirochete and causes lack of immunity. Spontaneous cure of the early disease does not destroy this early ample immunologic response, and the stronger the immunity the fewer the neurorecidives (meningitic or other). Local foci of spirochetes develop in the presence of a lesser immunity caused by insufficient treatment or when generalization is hindered. In other words, by means of insufficient early treatment or by a restriction of early immunity formation we have instead of the sterilisatio magna of Ehrlich, a sterilisatio fere completa. These views are combated by Matzenauer,[25] according to whom the severity and type of lesions are explainable on the basis of constitutional factors and individual susceptibility.

If immunity exists in syphilis, as is claimed because of the long period of incubation and invasion, the protracted latent period and various other phenomena, such as rare reinfection nonautoinoculability, we have as yet no scientific proof of this hypothesis.

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23. Brown, W. H., and Pearce, L.: J. Exper. M. **31**:749, 1920; **33**: (May 1) 1921; Am. J. Syphilis, **5**:1, 1921. In a series of rabbit experiments these authors have shown that in animals inoculated intratesticularly with virulent strains of spirochetes, those given one injection of arsphenamin and subsequently reinoculated after the disappearance of early clinical symptoms, developed typical reinfections in the shape of a second chancre, while the untreated infected controls failed to react a second time. By these experiments they attempt to prove that even in uncured syphilis, reinfection is possible if the resistance is lowered by insufficient "immunity" reaction.

24. Fraser, A. Reith: Am. J. Syphilis **5**: No. 2, 1921.

25. Matzenauer, R.: Wien. klin. Wchnschr. **32**:831, 1919.

The question of neurotropism of spirochetes may be merely glanced at. Some believe that certain strains have an elective affinity for the nervous system. In spite of the extensive animal work of Levaditi and Marie,[8] of numerous assertions of the rarity of nervous involvement among negroes, of cases of conjugal neurosyphilis, of the variation in morphologic appearances of certain types of spirochetes and of numerous instances of neurosyphilis in persons infected at the same source, we hold with Sicard,[26] Nonne[27] and a host of others that actual proof of the presence of special strains of the spirochete is as yet lacking.

Among our patients, and others have made similar observations, are persons infected at the same source, some of whom developed nervous lesions while their partners did not.

TREATMENT OF SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

Our own results in the eradication of clinical and biologic signs in the first year following infection with almost complete prevention of serious invasion of the central nervous system when treatment has been intensive enough, leads us to believe in the institution of therapy at the earliest possible moment and of such an intensity as is compatible with the patient's welfare. The proper care of a patient with syphilis of the nervous system demands that the physician be conversant with modern laboratory diagnosis, equipped to evaluate the biologic data, able to make a detailed neurologic examination and especially capable of reaching a logical and practical conclusion after digestion of all the evidence.

We strongly believe in the efficacy of combined treatment in most forms of syphilis of the central nervous system, with certain notable exceptions. We feel sure it is wise to adapt the treatment to the patient in the more chronic types of syphilis of the central nervous system rather than to carry out a stereotyped plan designed to eradicate biologic phenomena of the disease.

Let us realize that neurosyphilis cannot be judged or classified by the outcome of biologic reactions alone, but that of equal, often greater, importance are the clinical factors. Absolutely essential is a knowledge of the possibilities and limitations of various forms of therapy; also, a broad grasp of the patient as a complex human being with a psyche, not merely as a bundle of biologic phenomena to be made negative. We wish to protest against a form of therapy that often has a mortality of 20 per cent. (intracranial therapy) with merely promising results,

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26. Sicard, J. A.: Soc de neurol. de Paris, July 9-10, 1920; Presse méd. **28**:52, 1920; **28**:281, 1920; Médicine, Paris **2**:3 (Nov.) 1920.

27. Nonne: Syphilis und Nervensystem, Berlin, 1915.

and against all extra-hazardous and painful forms of therapy that do not offer more than a debatable chance for even prolongation of life, not to speak of cure.

Examinations of blood and spinal fluid in the so-called preclinical stage of syphilis of the central nervous system are of great importance. The case of the patient with fully developed neurosyphilis of any type is different. At this stage objective clinical signs are present and laboratory data must be looked on as corroborative, even though occasionally in the presence of dubious or merely suspicious clinical evidence, laboratory data may be the determining factors.

Treatment of Patients with Acute Cases.—In our previous paper we divided the cases clinically into three groups. In the first we placed all urgent cases with acute symptoms requiring immediate and intensive treatment, as well as all early cases of cerebrospinal syphilis of any type, early tabes and cases of general paresis. Toward these cases we have gradually assumed a more conservative attitude. We no longer follow the plan of Dreyfus, and we rarely give intravenous medication oftener than twice a week—more frequently only every five days until urgent symptoms are abolished, when the case is placed in Group 2, which includes the more chronic types of cerebrospinal syphilis, tabes and certain cases of general paresis. We rarely exceed 0.4 gm. of arsphenamin or its equivalent. The first injection we make minimal to avoid a Herxheimer reaction. In involvement of the medulla, optic or other cranial nerves, if time permits, we precede the injection of arsphenamin by one or two of a soluble mercurial (mercuric chlorid or succinimid) to prevent too intensive action by the arsenical. During the administration of any antisyphilitic in acute medullary involvement, the pulse and respiration are carefully guarded from the too intensive action of any spirocheticidal remedy. In the more chronic group, arsphenamin is given not oftener than once a week and in doses ranging from 0.25 to 0.4 gm., never higher. We believe that the spirocheticidal remedies are of no value if they exert their toxic action. We aim to get the tonic action of the arsenicals for chronic sufferers from specific nervous disease.

General Paresis.—As regards general paresis, we are convinced of the futility of intraspinal and intracranial therapy, which we unhesitatingly condemn. Well developed cases treated in groups for comparison did as well on one type of treatment as on another.[28] All eventually succumb. The tendency of dementia paralytica to sudden remissions, even intermissions, must always be borne in mind. Patients

28. Darling, I. A., and Weston, P.: Penn. M. J. **22** (March) 1919 (discussion). Jackson, J. A., and Pike, H. V.: Interpretation of Wassermann Reaction of Blood Serum in Mental Diseases, J. A. M. A. **76**:360 (Feb. 5) 1921.

with early cases frequently are astonishingly benefited by intravenous injections of arsphenamin, combined with injections or inunctions of mercurials, and exhibit prolonged remissions during which it is often quite impossible to detect any signs of mental aberration. The diagnosis of general paresis may offer exceptional difficulties even in the presence of positive biologic reactions in the blood and spinal fluid. In the absence of definite mental symptoms there has been a growing tendency to call these cases "serologic" paresis, paresis sine paresi.[29] The criteria of Darling[30] for true paresis are: speech disorders, pupillary changes, reflex changes, amnesia, quick shifting emotions, character changes and conduct slump. In the hope of a prolonged period of remission or that possibly what we believe to be paresis may prove to be a more hopeful cerebral syphilis, we practice persistent treatment of these cases.

Lumbar Puncture.—This brings up the question of lumbar puncture. We believe that on every patient with early syphilis lumbar puncture should be performed at least once, preferably at the end of the first year of treatment and earlier if indicated by clinical evidence of involvement of the nervous system. If the result is negative, it need not be repeated, if treatment is continued, until the third or fourth year. If then negative, in the absence of clinical signs of nervous syphilis, we do not repeat it(see plan). If positive, we do not repeat it oftener than once a year during the continuance of therapy. In latent cases or in late cases of syphilis with any suspicion of nervous or mental change, of course we perform lumbar puncture as a diagnostic measure. During treatment, we believe that a great deal of harm may be done by too frequent tappings. We are guided by the clinical symptoms and the blood reaction, reserving puncture for the completion of treatment. As therapeutic indications we are guided first by the clinical symptoms and objective signs, to a lesser degree by the outcome of the tests. We are not inclined to agree to the performance of puncture in the outpatient department or consulting room, but insist on at least one day's rest in bed immediately following tapping

Résumé of Treatment of Syphilis of the Central Nervous System.—Intraspinal Therapy: A review of the literature has shown a growing tendency in this country to restrict intraspinal treatment to very narrow limits which has been largely due to the efforts of Sachs and his associates. In Europe with few exceptions there has been a general abandonment of intraspinal methods.

Our former opinion, which we still hold, of the very limited value of intraspinal treatment was based on these facts:

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29. Southard and Solomon: Boston M. & S. J. **24**:1.
30. Darling, I. A.: Am. J. Syphilis **3**:35, 1919.

1. The amount of arsenic present in auto-arsphenamized serum was infinitesimal and of no spirocheticidal value.

2. There was no valid proof that arsphenamized serum was in itself spirocheticidal in vivo.

3. It was impossible to reinforce blood serum with more than an infinitesimal amount of arsphenamin.

4. The introduction directly into the cerebrospinal fluid of any of the arsenical preparations was often dangerous and occasionally exceedingly painful. When nontoxic quantities were used the remedy was impotent.

5. The pathologic changes in the various types of syphilis of the central nervous system are rarely only superficial, and are not reached by injection into the subarachnoid space.

6. The method was physiologically wrong. Nutrition of the cerebrospinal tissues is not afforded by the cerebrospinal fluid which serves as an hydrostatic agent for the suspension of the brain and cord and an avenue of excretion. Therefore, medication of the nervous tissues via the cerebrospinal fluid was impossible. Substances introduced into the cerebrospinal fluid are rapidly absorbed into the blood and rarely diffuse through the subarachnoid space. The direction of flow in the so-called perineuronal lymph space is away from the nervous tissue toward the cerebrospinal fluid. The pressure in the cerebral capillaries is greater than that of the cerebrospinal fluid so that fluid leaves the capillaries rich in material circulating in the blood, circulates in the pericapillary and perineuronal spaces, yielding nourishment and receiving waste matter, and finally leaves the tissues by the pericapillary and perivascular spaces to the subarachnoid cavities over the surface.

7. Arsenic injected intravenously regularly reached the cerebrospinal fluid in greater quantities than could possibly be injected intraspinally without damaging or destroying the nervous tissue. This is true also of mercury and iodids.

8. If the meninges are the seat of an acute or chronic inflammation, "antibodies" circulating in the blood stream reach the subarachnoid space. Intraspinal injections may serve to irritate the secreting mechanism and thus render it more permeable. Spinal drainage may act in a similar manner, as well as the reduction of fluid pressure.

If intraspinal therapy were a simple and harmless procedure, there would be little reason for an attempt to restrict its use. But we feel that the teaching of Fordyce and his followers is responsible for opening the door to a great deal of misguided and harmful effort due to lack of knowledge of the indications of this form of treatment, of failure to appreciate the true significance of the various biologic factors present in the disease, and above all, of insufficient experience in the various clinical manifestations of syphilis of the central nervous sys-

tem. In our previous paper we showed fallacies in the conclusions of these therapists and we believe that time has shown the narrow limits for this form of therapy.[31]

In optic atrophy intraspinal therapy is not contraindicated, but we feel that the disease, if not too far advanced, may be as satisfactorily rendered stationary or retarded in its progress by intravenous therapy. It is questionable whether serum administered in the lumbar region ever reaches the region of the base near the origin of the optic nerves. In properly regulated dosage if preceded by mercury and reinforced by iodids, arsphenamin has no harmful effect on the optic nerves.

If we restrict the use of intraspinal therapy, as Fordyce[32] has stated, to cases which after intensive treatment with arsphenamin, mercury and potassium iodid, show little or no improvement in symptoms, blood or fluid reactions, its field will be small indeed. It should by all means be a second rather than first choice in the treatment of neurosyphilis. Fordyce[13] now advocates its use especially in the early invasive period when the biologic reactions persist in spite of intensive therapy. On the one hand, Fordyce advocates the removal of these biologic reactions by intraspinal and combined therapy, and on the other, McDonagh,[33] Fraser,[24] and others advocate their retention as an evidence of reaction on the part of the neuraxis with protective antibody formation, a sign of good omen. As we have shown, the importance of these early reactions as regards actual involvement of the central nervous system has been misunderstood by Fordyce and others. McDonagh denies the efficacy of intraspinal therapy because in spite of it, late degenerative lesions may occur. In our opinion, the mere presence of spinal fluid changes in early syphilis does not call for intraspinal therapy, because these signs are transient and do not, in the vast majority of instances, necessarily signify nervous changes and only quite exceptionally resist treatment by arsphenamin intravenously and mercury. Here again we are adverse to the use of intraspinal therapy, except in the very rare instances in which a few years of proper treatment have left unchanged all the biologic reactions in the fluid.[34]

In the following plan we have briefly summarized our methods of procedure in all stages of neurosyphilis, as well as in early constitutional syphilis.

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31. What we believe these limits to be will be found in our article referred to in Footnote 1. Lack of space forbids their insertion here.

32. Fordyce, J. A.: Am. J. Syphilis **3**:341, 1919.

33. McDonagh, J. E. R.: Venereal Diseases, London, 1920.

34. In addition to the references given, the following may be of interest: Sachs, B.: J. A. M. A. **69**:681, 1917; Arch Neurol. & Psychiat. **1**:277-284, 1919. Dercum, F. X.: The Functions of the Cerebrospinal Fluid with a Special Consideration of Spinal Drainage and of Intraspinal Injections of Arsphenamized Serum, Arch. Neurol. & Psychiat. **3**:230 (March) 1920.

PLAN OF PROCEDURE IN SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

GROUP 1

Cases Requiring Immediate and Intensive Treatment:

Early cerebrospinal syphilis—at onset of or early in:

- Meningitis
- Meningomyelitis
- Meningoencephalitis
- Cerebral and spinal endarteritis (fresh insult)
- Optic neuritis—advancing
- Syphilitic epilepsy—earlier cases
- Pseudo tabes
- Ophthalmoplegias

Early tabes dorsalis—with acute symptoms, pains, crises, etc.

General paresis—with acute manifestations

Treatment—unless contraindicated:

1. Arsphenamin (or equivalent) 0.2 to 0.4 gm. every three to five days until symptoms are controlled, then 0.4 gm. every week—six to eight injections in each course. First course followed by treatment as in Group 2.
2. Mercury by deep intragluteal injections. Soluble salt every two to three days, alternating with above until symptoms demand less intensive treatment. Then follow injections by arsphenamin with weekly injections of insoluble preparations as in Group 2.
3. Iodids by mouth, if possible, rectally or intravenously otherwise.
4. Lumbar puncture for relief of coma, secondary hydrocephalus symptoms, root pains.

GROUP 2

More Chronic Cases—Later Stages of Group 1

Cases with persistent biologic reactions—general paresis:

1. Arsphenamin (or equivalent) 0.25 to 0.4 gm., once weekly or every ten days—six to eight injections followed by mercury.
2. Mercury, insoluble preparation every five days—or soluble preparation thrice weekly for two months or thirty rubbings.
3. Iodids by mouth, rectum or intravenously to point of tolerance for one month following mercury or alternating.
4. Sodium nucleinate intragluteal injections 0.3 to 1.0 gm. or more to induce febrile reaction—every 3 to 7 days—in general paresis and occasionally in resistant tabetic or spastic cases.
5. Reeducation—method of Maloney.
6. Symptomatic—baths, massage, medicinal for pain, bladder symptoms.

Repeat once or twice during the first year as indicated by benefit derived, the condition of the patient's excretory organs, morale, biologic reactions, etc. Subsequently yearly or more or less often, gradually tapering off. See Group 3.

GROUP 3

Late Cases of Tabes Dorsalis, Cerebrospinal Syphilis, Optic Atrophy, Spastic Paraplegia, and Cases of Group 2 after Three Years:

No attempt to influence biologic reactions, treatment mainly symptomatic and supportive.

1. Arsphenamin, 0.25 to 0.4 gm. every two weeks, not more than six injections in all; only if well tolerated.
2. Mercury by inunction or injection following No. 1.
3. Iodids by mouth, following No. 2.
4. Reeducation, method of Maloney.
5. Symptomatic, control of crises, bladder symptoms.

AUTHORS' METHODS OF TREATMENT IN EARLY CONSTITUTIONAL SYPHILIS

Treatment in Primary Syphilis:

Attempt at abortion of disease

Intensive intravenous arsphenamin therapy

Followed by mercury, rapid sterilization methods (Pollitzer, Scholtz)

Less intensive plans

Repetition of courses during first year.

Treatment of Secondary Syphilis—First Year:

(*a*) Attempts to eradicate symptoms.

(*b*) Attempts to eradicate biologic reactions: Arsphenamin intravenously every other day for three injections, then once weekly for five injections, followed by mercury twelve insoluble injections, twenty-four to thirty soluble injections. Rest from six to eight weeks, if symptomless, then arsphenamin weekly, for eight injections. Mercury, twelve insoluble injections, from twenty-four to thirty soluble injections. If Wassermann negative, rest for from two to three months. If Wassermann positive, repeat above treatment after from six to eight weeks' rest.

(*c*) Lumbar puncture: At the end of the first year, if the Wassermann reaction is negative and symptomless. At the end of second course, if the Wassermann reaction is positive or if there are symptoms of cerebrospinal involvement.

Treatment of Secondary Syphilis—Second Year:

(*a*) Symptomless—biologic reactions negative blood and cerebrospinal fluid—one course treatment—from four to six injections of arsphenamin, six injections of mercury.

(*b*) Symptomless—blood Wassermann reaction positive—cerebrospinal fluid negative. Two course treatment, six to eight injections of arsphenamin, twelve injections mercury insoluble, twenty-four to thirty injections of mercury soluble.

Treatment of Latent Syphilis:

After second year, Wassermann blood reaction positive.

1. Lumbar puncture to be advised. (*a*) Blood Wassermann reaction positive, cerebrospinal fluid negative. Treat as (*b*) above. (*b*) Cerebrospinal fluid positive—blood positive. Treat as Group 1 for one course, rest six to eight weeks, then arsphenamin weekly for six injections followed by mercury.
2. Repeat each year as indicated by symptoms and presence of biologic factors.

DISCUSSION

DR. WALTER F. SCHALLER, San Francisco: I have been interested in the pathogenesis of certain types of neurosyphilis and in the early diagnosis of this disease. My associate, Dr. Henry Mehrtens, has been occupied with studies of the cerebrospinal fluid, its pressure, circulation and absorption, and with the permeability of the meninges to drugs. Conjointly, along these lines, we believe to have found a justification for intraspinal treatment. In advanced cases, we can only hope for an occasional arrest and some symptomatic benefit. I believe that in certain forms of neurosyphilis, particularly in the meningeal form, such as tabes, we have obtained benefit when intravenous treatment has failed. In our experience the method is not dangerous although it requires a rigid aseptic technic. We give all our treatments in the hospital. There have been no more accidents in our series than in a corresponding number of arsphenamin injections alone. The intraspinal treatment includes, in addition to intravenous treatment, drainage and what additional benefit the application locally of specific medicines may have. The benefit has been both symptomatic and serologic. These patients return to us voluntarily, requesting further treatment, in spite of the physical discomfort and considerations of time and expense, because they are convinced that they have been helped.

DR. HARRY C. SOLOMON, Boston: Some patients do not respond well to intravenous treatment, but do respond very well to intraspinal therapy. These patients have developed essentially negative blood and spinal fluid findings and have improved their symptoms. We have treated our cases of neurosyphilis with intensive intravenous remedies for many months. In the last six months I have seen patients treated for three or four years with arsphenamin, mercury and iodids without effect, who after two, three, four or five intraspinal injections gave practically negative spinal fluid findings. There is some effect from intraspinal therapy, all theories to the contrary notwithstanding.

DR. HAROLD E. FOSTER, Boston: Several cases of primary optic atrophy, (syphilitic), have been treated for a number of years at the Massachusetts General Hospital according to the Swift-Ellis method administered intracisternally. The first patient selected for this method had been under intravenous arsphenamin treatment for about a year with slowly steadily contracting fields. In about four months from the first intracisternal treatment of arsphenamized serum the fields were of the same size as in the first examination. The process had apparently been checked. About six months later the fields were expanded slightly. One other patient who has been faithful to treatment was greatly benefited. Most of these optic atrophy cases are sent us by the oculists after the disease has progressed rather far. If the neurologic departments could get these cases in the early stages, this intensive treatment would obviate these extreme conditions, but many patients come to us when they are only able to distinguish daylight from darkness. Without exception we have had results with the intracisternal arsphenamized serum administration which were not obtained by the intravenous route.

DR. HENRY F. STOLL, Hartford, Conn.: I have had patients who continued taking intraspinal treatments even though they had a reaction after each treatment because they felt better than after the intravenous treatments. I do not think that intraspinal treatments should be given in all cases, but when the symptomatic and serologic improvement is not satisfactory, they should be resorted to. Patients showing the paretic curve with a positive Wassermann reaction in a small amount of spinal fluid should be given the advantage of both intravenous and intraspinal therapy as soon as they come under observation. The paretic curve does not signify paresis necessarily, but it probably is indicative of serious parenchymal involvement.

DR. DAVID J. KALISKI, New York: Our experience is based on the treatment of more than 500 cases by all methods of therapy. In the beginning when the Swift-Ellis method was first advocated we treated about 400 patients and some received as many as forty intraspinal injections. Occasionally, a patient

improved clinically and serologically, but patients also improved after lumbar puncture and normal serum intraspinally; in the vast majority of cases also if intravenous treatment alone was properly carried out. We do not mean arsphenamin intravenously alone, but the use of mercury and iodids in proper sequence and dosage with mental and mechanical therapy to increase the patient's morale and improve his general condition. These adjuncts are just as important factors as any one "kind" of treatment, and it also explains why some patients who are taken from one kind of treatment and put on another do well; it is because they are stimulated, buoyed up by new hope of cure, and their general condition improves. Intraspinal therapy should not be used in every case. That is what we desire to emphasize. As far as the improvement of optic atrophy after puncture of the cisterna magna is concerned, Dr. Foster cited only one case. This is hardly sufficient to do more than draw attention to the possibilities of the method. As to the paretic curve with the colloidal gold reaction, Dr. Tilney found, and we agree with him, that in about 155 cases the paretic curve did not indicate parenchymatous syphilis. The important point is that the paretic curve by itself does not mean paresis. The recital of a series of cases benefited by a given form of therapy is not a fair way of deciding the comparative merits of that form of treatment. Intraspinal therapy by itself, no matter how intensively applied, is not as efficacious, on the whole, as intravenous therapy, to say nothing of the comparative risks and inconvenience and expense to the patient. In the use of arsphenamin in syphilis of the nervous system we must avoid maximum dosage. Rarely use more than 0.4 gm. arsphenamin and 0.6 neo-arsphenamin. This applies to the intensive treatment in the early stages or in the later cases. It is all-important to avoid getting a toxic effect from too large or frequent dosage and to avoid any form of treatment that tends to disturb the morale of the patient with syphilis of the nervous system.

News and Comment

APPOINTMENT OF CHIEF OF DIVISION OF MENTAL HEALTH OF THE PENNSYLVANIA STATE DEPARTMENT OF PUBLIC WELFARE

Dr. William C. Sandy, Philadelphia, psychiatrist of the New York State Commission on Mental Defectives, has been appointed chief of the division of mental health of the Pennsylvania State Department of Public Welfare. The appointment became effective December 1.

Abstracts from Current Literature

RIGIDITY OF THE PUPILS AND THE CILIARY GANGLION FROM THE SPECIAL ROYAL RESEARCH CLINIC OF FLORENCE, DIRECTED BY PROF. E. TANZI. Cristoforo Rizzo, Riv. di patol. nerv. **25**:326 (April 4) 1921.

The pathologic anatomy of Argyll Robertson and fixed Rochon-Duvigneaud pupils is obscure, and the research work done in this connection is confusingly contradictory. Disturbances in various parts of the reflex arc for the pupillary movements have been held accountable for these signs. Déjerine places the lesion in the part connecting the anterior corpora quadrigemina with the third nerve nuclei; Mendel in the ganglia habenulae; Bechterew in the iridomotor nuclei of the third nerve; Mauthner in the third nerve roots; Wolf in the column of Goll in the medulla and Marina, Lafori and Sala place it in the ciliary ganglion. Marina found definite degenerative pathology in the ciliary ganglions and in the nerves of tabetic and paretic patients, who showed both the fixed and Argyll Robertson pupil. The roots of the ciliary ganglion were always found to be normal. The degenerated nerves were studied with Marchi, Weigert and the method of dissociation, the preparations being previously fixed with osmic acid. Marina is convinced that generally the degeneration begins in the ganglion cells of the ciliary ganglion, though it may less often begin in the nerves. Lafori also does not believe that these signs are due to lesions of the affective motor reflex arc (pain and emotional pupillary reflex pathways) but to sensorimotor reflex arcs subserving the light reflexes and accommodation-convergence reactions. Therefore the cause must be found in the ciliary ganglion and its peripheral neuron. André Thomas who did not, however, examine the ciliary nerve endings in the iris, reported three pathologic examinations with negative findings in the roots and ganglion of the ciliary nerve. This author used Cajal, osmic acid and picrocarmin stains.

Sala reported three observations in paretic patients with Argyll Robertson pupils, using the Cajal reduced silver nitrate stain. He believes that this sign is caused by the degenerative ciliary nerve and ganglion changes. The ciliary root was normal. In fixed pupils the roots, ganglion and nerves all showed more extensive changes.

Rizzo's studies were made of nine patients who had normal pupillary reactions and pupils, eight cases of paresis and one of tabes with Argyll Robertson pupils and one paretic with diminished light and accommodation reflexes and one paretic whose pupils reacted well.

The ciliary ganglion and nerves were carefully dissected out and treated with the following fixatives:

1. Ciaccio's liquid (aqueous solution of bichromate of potash 100, formaldehyd 20 and acetic acid 3 c.c.) for forty-eight hours and seven days of fixation in a 3 per cent. solution of bichromate of potash.
2. Lugaro's fixative (alcohol 100 c.c., nitric acid 5).
3. Alcohol, 95 per cent.
4. Ammoniacal alcohol (Cajal's formula).
5. Piridin.
6. Formaldehyd, 10 per cent.
7. Saturated solution of mercuric chlorid.

Most of the ganglions and ciliary nerves were treated with Ciaccio's liquid alone and then stained with sudan III or with Mann's mixture (aqueous solution of eosin and methylene blue). All the sections fixed with Ciaccio's fluid were stained also with fuchsin green (Alzheimer's solution No. 6) and with negrosin. Other staining methods were Cajal's silver stain, and those of Daddi, Spielmeyer, Bielschowsky and Biondi.

The ciliary nerves were sectioned in their entire course retrobulbar-intraocular and even to their terminations in muscle of Brücke, the iris and cornea.

The author warns against being deceived by staining artefacts caused by the various reagents and often interpreted as sclerosis, particularly of the large ganglion cells which predominate in the ciliary ganglion.

The author finds that the ganglion cells have a pigment which increases with age, such as occurs in sympathetic ganglion cells, but it is never as great as the amount of pigment seen in ganglion cells from the superior cervical ganglion. This pigment is best stained with Pappenheim's methyl pironin-green. The pigment is of two kinds, which in the author's tables are designated pigments A and B, and the differentiation is made according to the coloring given with any one of the several staining and fixing methods mentioned in the foregoing.

Pigment A is scanty and exclusively found in the small ganglion cells. It is present as small discrete granules sparsely spread throughout the cytoplasm, which is not rich in chromatic granules ordinarily. Only three or four cells in each section contain it. It occurs earlier than pigment B and does not tend to increase with age.

Pigment B is, however, abundantly present, occurs later and is exclusively found in the large ganglion cells. It occurs in closely packed granules, in clumps or in semilunar shapes in the most external part of the cytoplasm. This pigment increases with age or with the duration of the illness.

Pigment A of the small ciliary ganglion cells has many points in common with the pigment in the substantia nigra of Soemmering.

A short discussion of the minute anatomy of the ciliary ganglion, its preganglion and postganglion fibers, etc., precedes the author's description of the experimental methods used. The author felt that it was necessary to demonstrate that the staining methods used in the pathologic cases were sufficient and efficient. He therefore caused Wallerian degeneration of the ciliary nerves of a rabbit by cutting them just before they enter the sclera. Instantly this is followed by a fixed dilated pupil. The eye was enucleated forty-eight hours later and hardened in formaldehyd for several days. Sections of the cornea, Brücke's muscle, the iris and the sclera were cut and treated, the cornea with Bielschowsky's stain, the sclera, iris and Brücke's muscle with Daddi's and Spielmeyer's stain. Figures 19 and 20 of the author's article show the first phase of Wallerian degeneration in the myelin sheaths.

The ciliary ganglion of paretic patients with rigid pupils differs in no respect from the ganglion of normal persons (normal pupillary reactions). There was no diminution in the number of cells. The "piastrine" of Marina (spaces representing the residua of degenerated ganglion cells) are explained by the author as due to differences in the planes of the sections and not to degenerative processes. He stained various ganglions with Nissl's and Cajal's stains and proved this. The author reasons that if ganglion cells were gone they would be replaced by the interstitial connective tissue or by fibers from the capsule of the ganglions—but he found no such increase or replacement. The preganglionic ball-like endings about the ciliary ganglion cells in paretic

patients having rigid pupils did not show any abnormalities. There was no thickening or break in their continuity or forking of these fibers. No vacuolization was found in the ganglion cells of tabetic or paretic patients with fixed pupils.

The pigments mentioned by the author as occurring in the two types of ganglion cells was not affected in paretic patients with rigid pupils. Fat pigment, however, was increased. Contrary to Marina's findings, chromatolysis was seen rarely and only in the large ganglion cells. The amount of lipoproteins shown by the sudan III, Nissl and Lugaro stains was greatly increased in some cases and was probably an acute cellular reaction occurring in the last few days of life. These changes were most typically seen in an old man of 80 years dying of bronchopneumonia. His pupillary reactions were normal. The chromatolysis in this case occupied more than half the cells. In this case the lesion could be interpreted either as one of long duration and its existence compatible with the presence of a normally functioning ciliary ganglion, or it occurred during the last few days of life. The first cannot be the case because there was no loss in the number of cells. However, in the ganglion cells of paretic patients only 3.5 per cent. of the cells showed this chromatolysis; therefore the phenomenon of rigidity cannot be caused by these limited cell changes. These chromatolytic changes are seen also in toxi-infections and might be considered by some as part of an axonic reaction. Marked perivascular infiltration was noted in all the sections from paretic patients with fixed pupils.

All the roots of the ganglion were found perfectly normal in tabetic and paretic patients with fixed or Argyll Robertson pupils. The common third nerve root was also normal.

The small ciliary nerves showed no pathology either in the myelin sheaths or axis cylinders in cases showing either fixed or Argyll Robertson pupils. These nerves were sectioned and stained throughout their entire course even to the iris and cornea and not one pathologic fiber was found in cases showing fixed or Argyll Robertson pupils. Both the fibers of long and short ciliary nerves were normal. This disproves the supposition that the chromatolysis seen in certain ganglion cells mentioned in the foregoing is part of an axonic degeneration but is a toxi-infectious reaction too recent to cause axonal degeneration of a secondary nature.

Conclusions.—The larger amount of pigment seen in the large ganglion cells of the ciliary ganglion is the ordinary fat pigment. The pigment of the small cells is distinctive and like that in the substantia nigra. The former increases with age and the chronicity of the disease while the latter does not.

Normally the fibers of the long and short ciliary roots are much finer than the fibers of the nerves they leave. In the oculomotor root those fibers leading to the intrinsic eye muscles are morphologically different from those leading to the extrinsic eye muscles.

No cellular degenerations of great importance were found in paretic and tabetic patients with fixed or Argyll Robertson pupils. Such cell changes as were found were due to terminal infections.

No connective tissue reaction was noted either in the ganglion capsule or as replacements of degenerated or vanished cells.

The roots of the ciliary ganglion, the intraganglionic fibers and the short ciliary nerves of paretic and tabetic patients with pathologic pupils did not show degeneration.

The nerve plexus in the muscle of Brücke and the fibers in the iris and cornea were normal.

Perivascular lymphatic infiltration was a constant finding in the ciliary ganglions of paretic patients.

The same methods used to discover alterations in the ciliary nerves of paretic and tabetic patients with rigid pupils have disclosed experimentally produced Wallerian degenerations in the ciliary nerves of rabbits, in the thickness of the sclera, in the muscle of Brücke, in the iris and in the cornea.

A fair bibliography and twenty-seven good illustrations, uncolored however, are given.

OSNATO, New York.

CONCERNING MUSCULAR ATROPHY IN TABES DORSALIS. HERMAN LIPPMANN, Arch. f. Psychiat. u. Nervenkrankh. **63**:1, 1921.

This is a well written article in which the author takes up at length a very much neglected symptom in tabes, and makes the following general statements: 1. As a rule it attacks single muscles or a muscle group. 2. It usually occurs toward the end of a long drawn out disease; the muscles are then weak, anesthetic and atrophic. 3. When reaction of degeneration and fibrillary tremors are found the degenerative atrophy is certain, but when there is only a quantitative reduction in the electric excitability there is some doubt. 4. In general, the atrophy is usually limited to the lower extremities with a predilection for the lower part of the leg and then the foot. In the foot there is in tabes the characteristic "tabetic club foot" (pied bot tabetique; Joffroy). Occasionally the upper extremities are attacked, and then there is atrophy of the small finger muscles and the muscles of the thenar and hypothenar eminences. Of great importance is the atrophy of the shoulder girdle, giving the picture of an Aran-Duchenne type of atrophy, which is usually bilateral but not always equal. In occasional cases the trunk muscles, singly or in groups are involved. As a rule the atrophies as well as the arthropathies and the trophic disturbances of the skin occur in the later stages, but occasionally there is an exception. Charcot and Fournier have described a case in which the atrophy occurred in the preataxic stage. Lapinsky gives six cases of this in which paralysis with atrophy of various muscle groups began the disease. The classical cases with eye muscle symptoms are here briefly referred to.

The localization of the atrophy in the foot gives the characteristic "tabetic club foot" picture that is occasionally seen. The name "club foot," which Joffroy has given this condition, is perhaps not a happy one. It at first suggests a pes equinus. Other authors, as Leyden and Goldscheider, have used the words "club-foot deformity." The author suggests the term "tabetic pointed-curved foot." The author quotes this case: A woman aged 64, whose husband died of lung trouble, with previous negative family history, first complained of shooting pains in the legs at the age of 40. Nine years later after a severe menstrual bleeding, her legs became so weak that she was unable to walk, her gait being uncertain in the dark. Formication began in the extremities and the soles of the feet felt like fire. Two years afterward, both legs grew weak and she soon became bedridden.

The author examined the patient about ten years later and found the pupils small and of the Argyll Robertson type. The lower leg muscles on both sides showed atrophy and weakness. The greatest diameter of the calf was 25 cm. Both ankles were in a varus position; the feet were greatly distorted, the inner sides being more marked than the outer. The anterior part of the feet showed marked adduction. The first and second toes on both feet were

bent and abduction only slightly possible. Electric reaction gave a quantitative reduction—more on the left than on the right. There was a disturbance for touch and pain sense was lessened up to the knee. There was a band around the chest at the fifth and sixth ribs. Both legs and the left arm showed marked ataxia. The deep reflexes in all extremities were absent. Corneal, vomiting and abdominal reflexes were normal. The blood Wassermann reaction was positive, the spinal fluid Wassermann reaction negative. Roentgen-ray examination of the feet revealed distinct bone and joint changes.

The author considers first the anatomy of the tracts concerned in this case and then the pathology. He states that Lapinsky and others have found that section of the posterior root causes changes in the cells of Clark's column and in the nerve cells of the anterior horn, changes which in their essentials consist of a swelling of the bodies and loss of Nissl bodies. From this standpoint he explains the atrophy in tabes. In general two theories oppose each other: one by Déjerine and the other by Lapinsky. Déjerine thinks that the muscle atrophy of tabes depends on a neuritis of the peripheral nerves, which takes place very slowly, at the distal ends of the nerves, especially, and proceeds slowly to the anterior roots. These changes in the nerves indicate a proliferation of the endoneural and perineural connecting substance. The muscles which these nerves supply have thin, round fibers which in portions fall apart and are filled with pigment. The connective tissue in these muscles is increased and rich in nuclei—the fibers thin and few. Clinically there is atrophy of the extremities, fairly symmetrical. The spread indicates branching of the nerve trunk. Fibrillary twitchings are not observed; the reaction of degeneration is frequent. Déjerine is of the opinion, because of these findings, that tabes at times locates in the peripheral motor nerves. It has not as yet been proved that this is due solely to tabes. The clinical and pathologic findings are the same whether the neuritis is due to poisoning or to a mechanical injury. It has been known for a long time that in tabes, as in alcoholism, the motor nerves are vulnerable. Any toxin which a normal person can withstand may injure the nerves of an alcoholic or a tabetic person. Leyden, Remark, Möbius and others are of the opinion that in tabes there is a tendency to paralysis because through the degeneration of the posterior roots the impulses do not reach the anterior horn cells. The resistance of these cells is decreased, and they are easily injured.

A further objection to the theory of Déjerine is that in his studies he used the older methods of staining, and the author believes that the newer methods, especially the cell stains (Nissl's), are necessary to determine the entire picture, especially as regards the anterior horn cells. Lapinsky himself found no changes with the older staining methods, but he found them with the newer ones. The author also opposes the newer views of Déjerine that the changes in the anterior horn cells are the result of axonal degeneration and closes his discussion by stating that there are no good grounds for blaming these changes in tabes on a "tabetic" neuritis. Lapinsky is the leading exponent of the theory that the change is primarily in the anterior horn cells. With Nissl's stain he found in serial sections these changes in the cells: the cells were swollen, the Nissl bodies in part grouped around the nucleus. The nuclei were likewise swollen and altered in their shape and sometimes peripherally placed. Various peripheral nerves and muscles showed degenerative changes which had their origin in these cells. Clinically there were various differences from the neuritis of Déjerine. The localization of the atrophy does not indicate a distribution of the nerve trunks since some muscles were only in part atrophied, and in the same muscle normal bundles were

found near the involved ones. There was no symmetry in the affected muscles. The patient first noticed the diminution in size. Fibrillary twitchings were not observed.

Joffroy thinks the cause of the foot deformity was pressure of the bedcovers, while Déjerine and Marie believe the atrophy of the leg muscles, which results from the tabetic neuritis, to be the underlying cause. Marie also believes that trophic disturbances in the ligamentous apparatus of the joints result. According to the author, the pressure of the bed-clothes was not the cause. In tabes, as in all atrophies, we must look for the point of attack for the toxin in the anterior roots or in the anterior horn cells. This injury and, as a result, degeneration of the peripheral nerve, causes an atrophy of the leg muscles. Some parts of the muscles remain unimpaired because not all nerve fibers or cells are involved. Therefore there is only a quantitative reduction in the electrical excitability.

The author concludes that muscle atrophy in tabes occurs under these conditions: (1) accompanying disease; (2) as a result of general weakness and anesthesia of the muscles toward the end of the disease; (3) as a result of peripheral neuritis—brought out, not through the poison of tabes, but through other injurious processes; (4) as a result of the localization of the specific trouble of tabes in the nerve roots and anterior horn cells, whereby the motor impulses are more or less interrupted.

WINKELMAN, Philadelphia.

EPIDEMIC ENCEPHALITIS. G. MINGAZZINI, Ztschr. f. d. ges. Neurol. u. Psychiat., O. **63**:199, 1921.

"Non sunt multiplicanda entia sine necessitate" is the text of Mingazzini's excellent article on epidemic encephalitis. From his own clinical experience in more than a hundred cases, reinforced by necropsy findings in a large number, together with a well chosen and extensive review of the literature, he has constructed an article that is a credit to Italian investigators. He considers the disease a definite clinical entity that should not be clouded by the description of multitudinous "types," and he discountenances the terms "epidemic myoclonus" and "lethargic encephalitis." He divides the acute manifestations into three phases: the prodromal, the hyperkinetic and the lethargic phases. Often the sequence is observed, although the hyperkinetic phase may be absent, as was frequently the case in the earlier cases reported, or it may be the dominating feature of the individual case.

The author characterizes the lethargic phase as a pseudostupor and not a true coma, for in this period there are frequently hallucinations of sight and hearing. These are without the affect reaction which is so characteristic of alcoholic delirium, the patient pointing out the flies, mice, snakes, etc., as though they were of ordinary occurrence and not to be feared. Disorientation is usually pronounced. Occupational deliria are of common occurrence. The patient shows no interest in the future and has no insight. Catatonia, flexibilitas cerea and catalepsy are often encountered as motor anomalies of the lethargic phase.

The disease subsides by lysis. Where the hyperkinetic phase has been pronounced, the metamorphosis to the lethargic phase may be abrupt, but the recovery from the lethargy is usually slow and a post-encephalitic condition often remains that proves very obstinate and slow in disappearing. Among the most frequent of these sequelae, the author notes adynamia and apathy, which may be sufficiently pronounced to term them neurasthenia or even

melancholia. Often there are pains in the limbs, especially on movement, and muscular jerkings. Mannerisms may be observed. Another frequent sequel is agrypnia or inverted sleep rhythm. The parkinsonian syndrome is of frequent occurrence as a post-encephalitic complication, a large number of cases having been observed. Sometimes the disease begins with this syndrome, with a later onset of muscular jerkings and delirium.

An important statement of Mingazzini's concerning the occurrence of hemipareses in encephalitis is: "I have never seen a hemiparesis caused by a lesion of the internal capsule." He states that when weakness existed it was of slow onset and could always be explained by radiculitis. He lays much emphasis on the occurrence of radiculitis, both clinically and pathologically. Some of his arguments are worth stating: Pain and clonic jerkings are due to those diseases which exert pressure on the roots, and do not appear typically in poliomyelitis, syringomyelia or myelitis where the nervous substance is itself attacked. Herpes zoster has been observed, and exaggerated response to the galvanic current. Hiccup is said to be due to radicular irritation. Moreover, in sections of spinal cord there are to be seen extensive infiltrations of the spinal roots by lymphocytes, and the roots themselves are more or less compressed by them, as can be seen in the sections.

The author does not deny, however, that there is an inflammation of the gray matter of the spinal cord, for he refers to von Economo's findings, and he believes that paralysis of isolated muscles, such as the serratus anterior, may be due to this cause. Concerning paralysis of the ocular muscles also, the stand is taken that it is inflammation of the nucleus itself that causes the paralysis, and the author has prepared sections in which, for instance, the principal nucleus of the oculomotor group was pressed on by a large perivascular collar of lymphocytes. He states that he has never seen ophthalmoplegia in epidemic encephalitis. Rigidity of the pupils is a common affair, but subsides. The fact that one or the other of the divisions of the facial nerve may be affected speaks also for inflammation in the immediate vicinity of the facial nucleus.

The author takes up the consideration of the lethargy rather fully. Some investigators, he says, locate the sleep center in the floor of the aqueduct in close association with the eye muscle nuclei, so that oculomotor paralysis and somnolence often appear in common, but Mingazzini points out that tumors may involve this area and produce very definite paralysis without leading to lethargy. Fragnito, following Nonne, as a result of studies of the condition in lesions of this area, has termed the center the waking center, "Zentrum des Wachens," rather than "Schlafzentrum." Fragnito is against this conception, however, and places the center rather in the midbrain. Kennedy and Flexner believed that lesions of the thalamus, by blocking sensory impulses on their way to the higher centers, could cause somnolence, but here again, when much of the thalamus is destroyed, the author asserts that lethargy is not a characteristic finding. Barbara puts the whole question on an endocrine basis, there being excito-anabolic and excito-katabolic hormones which, secreted by the endocrine glands, act on the sympathetic system with resulting wakefulness or sleepiness. A disturbance of the balance will bring about disturbances of sleep mechanism. Mingazzini prefers, however, to base the symptoms on the pathologic findings, and in so doing follows the opinion that parenchymatous inflammation of the basal ganglions will give rise to somnolence. This is in accord with the frequency of other signs of basal ganglion disease, paralysis agitans, tremor, pain of a cerebral nature and indications of extrapyramidal disease.

For differential diagnosis he takes up only poliomyelitis and influenza, and shows clinically, experimentally and pathologically that epidemic encephalitis is different from either of these, although having some characteristics in common with both. The diagnosis can no longer rest, he states, on the triad—lethargy, fever and paralysis of the extra-ocular muscles—but the disease must be considered pathologically. It can affect either cranial nerves, or spinal nerves, especially the nerve roots; it can affect at one time the spinal cord substance, at another the brain, especially the brain-stem and basal ganglions, or it may localize about the ramifications of the basilar artery or the branches of the sylvian artery. If the pathology of the disease is understood, the diagnosis will be more satisfactory, and multiplication of types will be avoided.

In closing, concerning the treatment, he states that while some men have found intragluteal injections of milk or horse serum to be efficient against excitement, he has had the best results in treating the disease as a whole from the use of baths, leeches against the mastoid processes, small doses of bromids and milk diet. The subsequent apathy and "parkinsonoid" symptoms he manages with the best of hygienic treatment over the necessary period of months.

FREEMAN, Philadelphia.

A FEW REMARKS ON THE INSTITUTIONAL TREATMENT OF SCHIZOPHRENIA. A. REPOND, Schweiz. Arch. f. Neurol. u. Psychiat. **8**:190-199, 1921.

The possibility of instituting useful therapy in schizophrenia occurred to the author following a comment, by Bleuler, on the remark of a pupil, that so many of these patients had to be fed by means of a tube. He said, "It is your fault alone that these patients do not eat spontaneously." Following this rejoinder, the apathy of the attending personnel was so much improved that all but one of these schizophrenic patients were brought to eat spontaneously and developed a very hearty appetite within a few days. Since that time, whenever the condition of one of these patients not only ceases to improve, but tends to get worse, an attempt is made to discover what error on the part of the physicians and nurses was responsible for this change, and it is rare that some explanation, although it may be slight, cannot be found. He cites an example: A young catatonic woman with decidedly negativistic tendencies, but otherwise amenable to suggestions, permitted something to drop from her hand, apparently on purpose, and in very imperious tones demanded that it be picked up. This was not done. She immediately became absolutely hostile and until the date of her discharge, several months later, she did not speak. Another patient, the member of a titled Russian family, was offended because he had been addressed in a tone that seemed somewhat too familiar. Through the father, a plea was directed to the patient, and in this manner the attending personnel again ingratiated themselves, with marked amelioration of the patient's symptoms.

It has been a rather striking observation of the author's that in certain religious sanatoriums most of the schizophrenic patients fall asleep and become autistic. The writer looks on schizophrenia, from a practical standpoint, as resembling the psychic reactions of patients suffering from a cerebral injury, particularly when the post-traumatic manifestations are fluctuating, and when psychotherapy can accomplish such marked results. In the insidious forms this is not so well marked. In some of the latter cases Repond thinks he has

obtained excellent results by the administration of sodium nucleinate injections. He cites the following case: J. P., 21 years of age, was a sufferer from enuresis until 18 years of age. He normally drank well, and was very affectionate. Suddenly one day, he left his work and declared that he could no longer work because he was becoming too old. He refused to eat for the same reason and would not drink milk because it came from beasts with horns, which reminded him of the devil. In fact, at the institution, he refused all forms of nourishment, since he observed that all people who ate had a glassy stare, while his expression was frank and clear. He had to be tube fed, remained absolutely immobile with his eyes closed, refused to speak, and did not react to any form of excitation. Given 0.5 gm. (7.7 grains) of sodium nucleinate, his temperature rose, and the next day he ate spontaneously and answered questions. Thereafter he again became negativistic and refused to leave his bed until another injection of 1 gm. (15.4 grains) of sodium nucleinate was given. Following this his attitude again changed, so that the next day he was happy and enjoyed playing the harmonica. These injections were continued, and the patient has been at home for a period of four years, perfectly well.

In schizophrenics having melancholic syndromes the writer resorts to subcutaneous injections of oxygen. Alterations in the circulatory system are treated with injections of epinephrin.

Following Bleuler's advice, he thinks it important not to institute any form of therapy that may be too vexatious to a negativistic patient. The application of therapy must be undertaken with tact, with a definite purpose in mind, and at the proper time. He believes that this can be done only when the patient and the physician have established a friendly relationship; then it is possible to decide on measures to be adopted. If no indication can be found, it is necessary to employ certain procedures as a test, for example, suddenly taking the patient out of a continuous bath or out of an isolated room for a walk in the open. The desires of the patient must be taken into consideration. He cites an interesting case in this connection: The patient was an old nurse who had for two months been in an acute catatonic crisis. She isolated herself, cried and assumed very threatening attitudes toward the staff. One day, however, there was immediate need for a nurse to serve at night. As an experiment, the patient was abruptly asked whether she would accept this position. The reaction was intense and the patient immediately became frank, thanked the physician, and that night, under observation, rendered excellent service. This surveillance, however, was not necessary, as further experience showed. She was devoted in her work and made interesting observations. In the morning, however, just as soon as she removed her habiliment she suddenly became catatonic, demanded that she be isolated or else hid herself, and would not sleep except by means of narcotics. Toward nightfall she became calm and at the prescribed time resumed her occupation. This was continued for a considerable period of time.

The task, accordingly, is to find a favorable opportunity to break into the autism of the schizophrenic. The best way of doing this is by instituting some form of work. This must not be too mechanical or schematic but must represent an occupation to which the individuality of the patient is adapted. It is also important that the rules of a sanatorium be sufficiently elastic not to impose too great a restriction on the patient.

It seems paradoxic that the dismissal of the patient from the institution can sometimes be used as a therapeutic measure. Repond has seen some

excellent results follow immediately after dismissal, even when carried out against the wishes of the relatives. He thinks that this is important, particularly as it applies to the larger state institutions in which residence represents little more than room and board at public expense.

WOLTMAN, Rochester, Minn.

FURTHER COMMUNICATION CONCERNING THE PECULIAR INCLUSIONS IN THE GANGLION CELLS OF CORPORA AMYLACEA IN AN UNDOUBTED CASE OF PARAMYOCLONUS MULTIPLEX. A. WESTPHAL and F. SIOLI, Arch. f. Psychiat. u. Nervenkrankh. **63**: 215, 1921.

The case on which this article is based was reported in brief by Westphal a short time ago (*Arch. f. Psychiat.* **60**: Nos. 2 and 3). A boy, aged 18 years, with a negative family history, said to have had his first convulsion at the age of 10, some time after an attack of scarlet fever began to have myoclonic spasms and epileptic attacks which attained marked intensity and severity. The myoclonic spasms involved the entire voluntary body musculature, were quick and lightning like, with disturbances of motion. There was gradual mental deterioration. He soon became blind and deaf and the amblyopia increased without positive findings in the optic apparatus. Sometimes he had visual hallucinations. At times the pupils did not react; at other times they reacted sluggishly to light, the reaction being either one-sided or bilateral. There was likewise fluctuation in the skin and deep reflexes. The Babinski and Oppenheim reflexes were also variable, being weak or absent. These changes were evidently connected with the epileptic attacks. The gait was reeling, uncertain and cerebellar. His manner of speaking was changeable, being slow and scanning at times. All laboratory work was negative. In the last stages there was undoubted myoclonic reaction. Death occurred, nine years after the appearance of the first symptoms, in extreme dementia with difficult speech.

The brain weighed 1,330 gm. and no gross changes were noted. The brain was cut through the middle, the left half placed in 96 per cent. alcohol and the right half in liquor formaldehydi. After fixation there was seen in the left cerebellum a peculiar localized change in the medullary layer beginning in the region of the dentate nucleus. The white matter was colored a yellowish-brown without disturbance in the consistency. A detailed and complete study was made of the various parts of the brain by every conceivable stain, and this summary is given of the findings: "All parts of the central nervous system were studied and were not found atrophied. In the vessels was found neither infiltration nor disease. In the entire brain there was a slight increase of glia cells and fibers, especially in the outer layers, and some pigment was found in the vessel sheaths. Lipoid was seen in the ganglion cells. The case is distinguished by peculiar deposits, especially in the gray matter, and these can be divided into two groups: (1) corpora amylacea, most of which lie within the ganglion cells, and (2) glycogen-like granules, most of which lie extracellular. These deposits are found all over, but are more numerous in certain places. The ganglion cells are altered in the entire brain. These changes are of various types and in portions very severe. The deposits and the severe ganglion cell disease are most marked in the thalamus, nucleus ruber and nucleus dentatus; the difference is so manifest that one must talk of an elective intense disease of these gray nuclei."

Then follows a classical discussion of the mechanism of these deposits and their exact composition with no definite conclusions. The authors discuss the conditions in which foreign material appears in the cells, such as lipoid pigment in senility, etc. They state that it is not clear whether the inclusions are primary and begin the cell disease, or whether they are secondary.

WINKELMAN, Philadelphia.

SYMPATHETIC PAINS. J. TINEL, Presse méd. **29**:263 (April 2) 1921.

War injuries have called attention to various traumatic causalgias and similar pains of sympathetic origin. Such pains are localized at the cutaneous end of a nerve, rather than along its course, and are much more severe over the peripheral part of a limb than at the wound level. Superficial irritation, especially dryness of the skin and contact with the air, is particularly distressing. Deep pressure is less so. Typically characteristic are the painful crises aroused by any lively emotion or sudden sensory stimulus.

Ordinarily there is no anesthesia, but rather hyperesthesia and no paralysis or change in electrical reactions. Trophic vasomotor signs are marked; usually vasodilatation—hot, red, and profusely sweating skin—is noted; rarely, vasoconstriction—cold, white, dry and squamous skin.

Any mixed or sensory nerve may be affected. The median and the internal popliteal are most liable; the ulnar and the sciatic trunk less so.

The disproportion between an insignificant lesion and intense symptoms is often striking. The hypothesis of their sympathetic origin rests on the following evidence: (1) the nerves most often affected are richest in sympathetic fibers; (2) the pains have a special sympathetic character comparable to the sensation of numbness after cold, especially to that of warming after numbness; (3) the distribution of the sympathetic symptoms is often quite different from the distribution of the nerve trunk affected, and symptoms vary from day to day ; (4) persistence of causalgia is possible after section of the nerve apparently involved, while cures have been effected by section of the nerve below the wound or by periarterial sympathectomy; (5) referred pains often appear, extending either to other homolateral nerve distributions or to the same contralateral distribution, or to the whole of one side of the body.

Tinel concludes that a wound (generally slight) of a nerve trunk, either by the pain that it causes or by the irritation of sympathetic fibers that the peripheral nerve contains, can provoke a state of erethism or excitability of the corresponding sympathetic centers. This brings on not only the pains but also the vasomotor and secretory disturbances, and the extreme irritability of sympathetic terminations in the vicinity. Irritation of sympathetic centers can then progress to neighboring centers.

Symptoms following the irritation of wounds resemble those appearing in several different nontraumatic cases, which are fully described and compared. These symptoms are found in common:

1. Irritating rather than ordinarily painful sensations, often of agonizing degree and persistence; tension, fulness, numbness, intolerable heat, violent burning, momentarily helped by cold water, and associated often with arterial throbbing.

2. Absent disturbances of motion and sensation.

3. Irregular appearance of the sympathetic pains in paroxysmal exacerbations, and precipitated or exaggerated by emotion; also their progressive attenuation by physical and especially mental rest.

4. Progression and exaggeration of the pains in the absence of rest or satisfactory treatment.

5. Their limitation to predisposed subjects with irritable sympathetic nerve systems. Anxious states often accompany the pains either as cause or effect.

The mechanism of this sympathetic disorder is obscure even when a demonstrable point of irritation is present. Treatment is similarly unsatisfactory; surgery is apt to make matters worse; physiotherapy is unsuccessful, and of all the drugs that have been recommended Tinel has only found two that are at least occasionally of value—potassium iodid and calcium chlorid. He obtains best results from a combination of rest, mental hygiene and persuasion.

HUDDLESON, New York.

CONTRIBUTION TO PATHOLOGY OF THE THYMUS GLAND. 1. CHANGES IN GROWTH FOLLOWING THYMECTOMY. EUGEN BIRCHER, Schweiz. Arch. f. Neurol. u. Psychiat. **8**:208-214, 1921.

Next to the thyroid the thymus gland has the greatest influence on the growth of bones. Basch was the first who attempted to remove the thymus experimentally and to note the results. The changes noted were confined mostly to the osseous system. The bones became generally much more pliable and frangible; the gait was awkward; the psychic reaction changed; callous formation following fracture was impaired. The long bones were most markedly involved, the ribs and hands less so. The epiphyseal lines were widened and irregular.

Matti and Klose noted relatively greater involvement in the posterior extremities, the formation of a rosary and spontaneous fractures following thymectomy in dogs. Matti attempted to identify these changes with those seen in rickets.

We know comparatively little of the after-effects of thymectomy in man. The attempt of Klose to establish a picture of idiotia or cachexia thymipriva has not as yet been successful.

The thymus is one of the few glands of internal secretion undergoing involution in post-fetal life. About the ninth month of life the thymus is firm and filled with secretion; from the third to the fourteenth year it is stationary in size; however, there is an increase of connective tissue and the secretions diminish quantitatively; from 25 to 50 years of age it diminishes in weight, and the secretion also becomes impaired qualitatively; complete disappearance usually does not take place.

There can now be little question as to the existence of the clinical picture of tracheostenosis thymica, the main symptom of which is dyspnea. Up to the present time several hundred such cases have been reported; however, only fifty patients have been operated on. The writer has had occasion to operate on ten such patients. In eight he had the privilege of examining the patients after intervals up to nine years. Complete removal of the thymus was not undertaken; indeed, it would be quite difficult on account of the aberrant thymus bodies. A subsequent examination showed rather convincing evidence of a delay in the appearance of the centers of ossification, allowing two years as the normal range of variation. This delay in some cases was found to be as great as six years.

While some of the cretinism seen in certain districts of Switzerland cannot be attributed to the disturbance of the thyroid gland alone, but, according to Holmgren, must represent an anthropologic variation, the latter interpretation does not seem to apply to these cases.

Bircher states that it is by no means necessary to remove the entire gland, but that removal of one fourth of it is enough to produce these changes. The diaphyseal lines in all cases were normal so that the writer does not feel that these changes can be explained on a rachitic basis. In two cases there was a deformity in the navicular and calcaneus bones of the feet, in which the former were seen to be small, irregular, and ragged in outline, while the latter were large but also irregular in contour. In these two cases there was a psychic factor, as evidenced by apathy, indifference and difficult progress in school.

In concluding, Bircher indicates that thymectomy must not be lightly considered and that radiation of the whole organ by means of the roentgen ray is to be discouraged.

WOLTMAN, Rochester, Minn.

ETIOLOGY AND THERAPY OF MULTIPLE SCLEROSIS. FRITZ KALBERLAH, Berl. klin. Wchnschr. **58**:963 (Aug. 15) 1921.

According to Kalberlah, multiple sclerosis is the most common organic nervous disease with the exception of tabes and general paralysis. The apparent increase of the disease may be attributed to earlier diagnosis and to diagnosis on relatively fewer symptoms, such as transient eye palsies, passing scotomas, etc., in the absence of syphilis.

Strümpell's theories as to etiology have been abandoned. At present, following the work of Siemerling, Spielmeyer and others, the theory that the disease is due to an inflammatory change seems well founded. The pathologic picture is that of a hyperemia, infiltration of vessel walls and adjacent tissues with plasma cells and later destruction of the axis cylinders, disappearance of myelin sheaths and of ganglion cells, with focal changes in the meninges. Finally there is a glial proliferation with atrophy and shrinking of the brain and cord.

It is this pathologic picture which stimulated a search for an exogenous toxin. The work of Kuhn and Steiner in 1917, in which the disease was transmitted to rabbits and guinea-pigs and a spirochete isolated from the blood and liver of these animals, seemed to fulfil the prophesies of many.

It has been disappointing that other investigators have been unable to repeat the work of Kuhn and Steiner. The author, in 1920, obtained practically the same results as the earlier investigators. In support of his own findings he states that Siemerling, Büscher, and others, have been able to demonstrate a spirochete in fresh foci from cases of multiple sclerosis, but were unable to stain the organism.

The writer believes that the lack of corroboration makes it necessary to be cautious in drawing conclusions. He mentions the difficulty of staining spirochetes in the brain and the numerous errors made in calling nerve tissue spirochetes. Kalberlah believes that one is justified in saying that multiple sclerosis is in all probability an infectious disease due to a spirochete.

Since 1918 Kalberlah has used silver arsphenamin in the treatment of thirty-six patients suffering with multiple sclerosis. The results have been favorable though no cures have been effected. Patients with mental symptoms have shown the least improvement. Similar results have been reported by other observers in Germany. The writer does not think that arsphenamin is the ideal remedy, but up to date it presents the best agent, having in the arsphenamin an antispirochetal effect and in the silver a special affinity for nerve tissue. The method of use is described, being essentially the same as that employed in tabes. Special emphasis is placed on continued treatment.

Because of the pathology of multiple sclerosis it is of the utmost importance to make an early diagnosis as in chronic cases with glial changes little can be hoped for. However, in these cases treatment should also be instituted as it is of value in warding off new foci, even if the old lesions cannot be affected. In the chronic cases little may be accomplished because of the extensive destruction which has already taken place.

In conclusion the writer insists that once the treatment with silver arsphenamin has been started, it should be continued intermittently for years, as in tabes, even though the improvement is slight, and it is essential that one does not become discouraged quickly.

MOERSCH, Rochester, Minn.

HYPOPHYSEAL SYNDROME OCCURRING WITH TUMOR OF THE THIRD VENTRICLE WITHOUT HYPOPHYSEAL INVOLVEMENT.

P. LEREBOULLET, J. MOUZON and J. CATHALA, Rev. neurol. **28**:154 (Feb.) 1921.

The protocol in the authors' case may be summarized thus: The family history was negative except for evidence of neurosyphilis (type unspecified) in the father. The patient was a young man, aged 26, presenting what was considered clinically as a typical Fröhlich syndrome: segmental adiposity, hypotrichosis, delicate texture of the skin, genu valgum and gonadal hypoplasia, with apparent absence of libido. In addition, severe cephalgia, attacks of vomiting, and diplopia, with diminished visual acuity, were reported, dating from the age of 17. At 20, vision was found to have been completely lost in the right eye and temporal hemianopsia was determined in the left. Roentgenologic examination of the sella turcica was negative until the age of 25 when evidence of excessive calcification was found in the region of the clinoids. Neuropsychiatric examination at this time was apparently negative, and no further physical deviation was observed. Examination of the spinal fluid showed evidence of slight lymphocytosis (15) and increased solids, although these abnormalities were not uniformly present on subsequent examinations. On the occasion of the last spinal fluid examination (just before death) the Wassermann reaction was found to be positive, although the blood Wassermann reaction was negative; no mention is made of the colloidal gold reaction or of the character of previous blood and fluid Wassermann reactions.

On final presentation at the authors' clinic (when the patient was 26 years of age), there was reported definite exacerbation of the patient's complaints as noted: severe headache, attacks of vomiting, impaired vision and extreme weakness. Shortly following this, bradycardia was observed, and the patient passed into a state of progressively more marked somnolence, which was characterized, at times, by delirium and which terminated finally in death, having been immediately preceded by coma, marked by mydriasis, strabismus, a bilateral Babinski sign, partial rigidity and incontinence.

At necropsy there was found a cystic papillomatous epithelioma, showing areas of calcification and apparently primary in the third ventricle, but extending basally into the interpeduncular space, encroaching on the optic chiasm anteriorly, compressing the infundibular stalk and extensively involving the mamillary tubercles and the gray matter of the anterior perforated space, with some displacement of the cerebral peduncles posteriorly. The pituitary body, aside from the compression of the infundibular stalk, as described, showed no evidence of pathologic alteration, nor was there indication of disturbance histopathologically in any of the other endocrine bodies with the exception, perhaps, of a certain degree of orchitic sclerosis.

In view of the very definite picture of the Fröhlich syndrome presented clinically, the authors are struck by the apparent absence of endocrine involvement, particularly hypophyseal, and suggest, in explanation, the possible relation of the determined compression of the infundibular stalk. They also feel that their case may serve to throw some light on the hypothesis recently advanced as to the possible directing influence of the tuber cinereum and the basal gray matter (involved in this case) on sexual development and the production of somnolent or torporous states. Of especial interest in connection with the present case is the report recently made by Claude and Schoeffer (Tumor of the Third Ventricle with Compression of the Hypophysis but Without Hypophyseal Syndrome, *Rev. neurol.* **28**:25 [Jan.] 1921) of a large third ventricle tumor involving the sella turcica, with severe pressure atrophy of the entire pituitary body, particularly the posterior lobe, and inferentially the infundibular stalk, unattended, however, by any clinical evidence of the so-called hypophyseal syndrome.

RAPHAEL, Ann Arbor, Mich.

COMPULSION AND DEPRESSION, A CLINICAL STUDY. BECK, Monatschr. f. Neurol. u. Psychiat. **48**:273 (Dec.) 1920.

According to Westphal, the criteria of a compulsive idea are that it develop in the mind of a person whose intellect is unimpaired; that it be not determined by feeling or emotion; that it force itself into the consciousness of the person against his will, and remain in spite of his efforts to displace it; that it prevent the normal flow of ideas; that it be appreciated by the patient as something foreign to himself. Four points are essential to the fulfilment of Westphal's definition:

1. The intellect must be intact.
2. The compulsive thought cannot be displaced and must impede the normal flow of ideas.
3. The thought must not be caused by affective factors.
4. The patient must have insight.

The author accepts the first two of Westphal's criteria, and devotes his paper to an attempt to demonstrate by case reports that affectivity plays a definite rôle in compulsion processes and that insight is not always complete.

Whereas there are undoubted cases which come entirely within the scope of Westphal's definition, there are many more in which a definite relationship—even a causal relationship—may be shown between compulsion process and emotional disturbance. So closely is the whole group of compulsion neuroses related to the manic-depressive group that difficulties of diagnosis are constantly cropping up. All transition types occur, and the author, in common with many others, seriously doubts the feasibility of making a differential diagnosis between manic-depressive insanity and psychesthenia.

Summarizing, he states that the compulsion neuroses may occur independently; but in both the acute and chronic forms they may be intimately associated with manic-depressive insanity; compulsion neuroses may run the same type of course as manic-depressive insanity, and in many cases emotion is the cause of the development or progress of the symptoms. In some cases, insight into the content of the compulsive thoughts is lost. Differential diagnosis between the two groups leads to numerous difficulties—both in acute phases and in the constitutional types. The more intensively one studies such persons, the more firmly is one convinced that a grouping into the ordinary diseases is impossible. We may label such a patient as having a degenerative

constitution, or as a psychasthenic or manic-depressive—but this means little; it is only by an intimate study of each patient that we can understand him, and it is that understanding and not classification that counts. The author then gives a table showing the relationship between the various types of disturbed emotional life.

SELLING, Portland, Ore.

PARALYSIS IN CHILDREN DUE TO THE BITE OF WOOD-TICKS. P. D. MCCORNACK, J. A. M. A. **77**:260 (July 23) 1921.

The author reviews the literature dealing with paralysis in children and adults resulting from the bite of wood-ticks (*Dermacentor venustus*). This animal is also responsible for the transmission of Rocky Mountain spotted fever. Its bite causes "paresis, paralysis and, in some cases, death." A similar syndrome has been found in sheep to which it has been found attached. Adults are rarely affected. It is interesting that among sheep the yearlings alone become ill; the older sheep apparently acquire immunity.

The tick season may begin "as early as February and last until August." Southern British Columbia, eastern Washington and Oregon, Idaho and Montana are the usual localities. Cases are also reported from Cape Colony and Australia.

Both in sheep and man, more than one tick may be found, but the increase in number does not seem to influence the severity of the disease. It has been produced experimentally in sheep by the attachment of the tick, but the disease has not been transmitted from a sick animal to a well animal by inoculation of material from the central nervous system. The paralysis may clear up before removal of the tick. No definite pathology except slight congestion of the brain and spinal cord has been found in animals dying from this form of paralysis.

"The symptoms are usually characterized by the sudden onset, in a previously healthy child, of weakness in the muscles of the extremities, staggering, difficulty in standing, and disinclination to active play, rapidly followed, in a few hours, by more or less complete motor paralysis. The child is often unable to stand, hold the head erect, or feed himself, although consciousness is not impaired. The onset may be accompanied with convulsions. The incubation period in children is doubtful, but, by experiments in animals, it is found to be from six to seven days after the ticks are attached. Rapid pulse and a slight rise of temperature have been noted. A large engorged wood-tick is found somewhere on the body, most often in the scalp, occasionally in the external ear, the axilla and other protected areas."

The prognosis is good when the tick is removed early. The patient's recovery may be looked for in forty-eight hours. When the paralysis has become extensive death may follow from respiratory failure. The disease may be confused with polio-encephalo-myelitis, meningitis or an acute intestinal intoxication.

In one case a blood examination revealed eosinophilia. The treatment consists in removal of the tick. Great care must be exercised to remove its head.

KRAUS, New York.

A REVIEW OF A YEAR'S SERIES OF INTRACRANIAL TUMORS. C. E. LOCKE, JR., Arch. Surg. **3**:560 (Nov.) 1921.

In this review, Locke reports 255 cases with the presumptive diagnosis of brain tumor that occurred in Cushing's clinic during twelve months. Of

this number, 107 were verified brain tumors, 60 unverified, 56 brain tumor suspects, and 32 belonged to the nontumor group. There were in all 180 operations performed and 20 fatalities. This is a creditable showing, and reports such as these are useful in that they serve to strengthen the growing conviction that the results of such surgery are already within reasonable limits of expectation. Such reports also serve to strengthen the belief that cranial exploration should be resorted to more frequently. There is no more reason for hesitating to carry out an exploration of the brain or spinal cord on justifiable symptoms than there is to hesitate to explore the abdomen, provided the exploration is done by one qualified to do it. Forty-two and one-half per cent. of the total number of tumors belonged to the glioma group, and, as the author states, these figures are in keeping with the general idea that gliomas are the most frequent tumors of the brain, constituting from 40 to 50 per cent. of all intracranial tumors. What is not so generally known, however, or rather, appreciated, is the fact that much can be done by surgery in this group to prolong life. Occasionally, even a solid glioma can be removed. Gliomatous cysts can be drained, and, if in a favorable location, sometimes removed. There were sixteen gliomatous cysts out of a total of forty-five gliomas in this series. The cerebellum was the usual location and 73 per cent. occurred in children. This report is well worth reading in the original.

RODMAN, Philadelphia.

EARLY CONTRACTURES OF REFLEX ORIGIN—THE HORMETONIC SYNDROME. S. DAVIDENKOF, Rev. Neurol. **27**:9 (Jan.) 1920.

Davidenkof proposes the term hormetonic (ἡ ὁρμή -stroke; ὁ τόνος -tension) syndrome, in designation of the early tonic hyperkinesias frequently noted in the paralytic field in encephalitic disturbance, particularly where associated with coma, such as the various cerebrovascular accidents, encephalomalacia, brain abscess, cranial injuries, attended by crushing of cerebral tissue, and the encephalitides.

This syndrome, which may vary greatly in degree, and which has been regarded by most observers as a manifestation of pyramidal irritation, becomes evident as a rule, from one to three days after the onset of paralysis. It is characterized primarily by intermittent muscular rigidity in the zone of paralysis (occasionally other areas may become involved), marked by fixed attitudes or contracture-states, interrupted at intervals by involuntary tonic movements. The reflex of defense is definitely exaggerated, and is characterized by the same movement-components as compose the involuntary tonic spasms. The condition is essentially transitory, and tends to disappear gradually in parallel with the abatement of coma, although in rapidly progressive cases it is generally found to persist until death occurs. These early or "precocious" contracture-states may, or may not, be later succeeded by permanent paralytic fixations.

RAPHAEL, Ann Arbor, Mich.

THE ACHIEVEMENTS AND LIMITATIONS OF NEUROLOGIC SURGERY. CHARLES H. FRAZIER, Arch. Surg. **3**:543 (Nov.) 1921.

In this address, Dr. Frazier expresses his views on neurologic surgery, which have grown out of his large experience. He is one of the very few whose experience is sufficiently great in this line of work always to justify a respectful hearing. When coupled to this is the well-known discrimina-

tion which Dr. Frazier brings to his work, one can safely follow his lead. The address discusses in turn certain facts in the comparatively recent history of this specialty in surgery, types of tumor and their treatment, pseudotumors (so-called), subtemporal decompression, major trigeminal neuralgias, surgery of the pituitary body, tumors of the spinal cord, surgery of the spinal roots, chordotomy for the relief of pain and certain matters of technic. It was interesting to the reviewer to read Dr. Frazier's opinion of the intravenous use of saturated salt solution to control increased intracranial pressure (Weed and McKibben), as he had the impression that this was already proving to be a useful procedure. In the light of the opinion expressed in this review, however, it apparently is a procedure to be used with caution. All of those interested in neurologic surgery should read this address of Dr. Frazier's, which so authoritatively discusses the subjects mentioned.

RODMAN, Philadelphia.

NEW OBSERVATION CONCERNING THE PATHOGENY OF A NEW SYMPTOM OF MENINGITIS. G. R. LAFORA, Rev. de méd. **38**:366, (June) 1921.

In 1915, the writer first pointed out a symptom in meningitis, not previously described, namely, nasal itching, dependent, in his opinion, on irritation of the trigeminal branches or of its ganglion. The nasal mucous membrane has sensory ennervation from four trigeminal branches: (1) an internal nasal nerve, branch of the ophthalmic, (2) sphenophalatine nerves, branches from Meckle's ganglion, (3) posterior nasal nerve, a branch from the posterior palatal, and (4) the ptergopalatine, a posterior branch from Meckel's ganglion.

Basilar meningitis often causes an inflammatory encasement and toxic irritation of the gasserian ganglions, and this can extend along the nerves mentioned. It is an irritation phenomenon akin to the development of convulsions in cases of helminthiasis.

The patient reacts to the itching sensation by persistent scratching of the nose, and this behavior in meningitic cases may continue even into the period of semistupor. It is not an exclusive symptom of cerebrospinal meningitis but is seen in all types, including tuberculous meningitis and the post-otitic.

The writer reports a case in detail in which the symptom was present unilaterally.

HUDDLESON, New York.

TUMOR OF THE THIRD VENTRICLE WITH COMPRESSION OF THE HYPOPHYSIS BUT WITHOUT A HYPOPHYSEAL SYNDROME. H. CLAUDE and H. SCHOEFFER, Rev. Neurol. **28**:25 (Jan.) 1921.

The protocol in Claude and Schoeffer's case may be summed up as follows: The patient, a toolmaker, aged 29, entered the authors' service complaining of severe headache, vomiting, failing vision, somnolence and general asthenia, dating back ten months, associated with a gain of 22 pounds in weight (apparently nonsegmental), sexual impotence and character change, marked particularly by extreme irritability and jealousy. Examination showed marked somnolence, diminished visual acuity with bilateral papilledema, retardation in the kinetic field, absence of abdominal reflex response, slight lateral nystagmus and a pulse rate of 60. Examination of the spinal fluid proved negative. Seventeen hours after admission the patient passed into a state of coma which lasted approximately an hour, in which were noted loss of corneal

and pupillary response, anisocoria, rigidity over the entire right side and urinary incontinence. On the fifth day the Babinski sign was secured on the left and the pulse rate was found to have been reduced to 52 with marked deepening of the patient's torpor, associated with Cheyne-Stokes respiration. Decompression was performed the following day, the patient dying, however, some hours later without regaining consciousness.

At necropsy examination the third and lateral ventricles were found to be widely dilated and the former to contain a cyst about the size of a pigeon's egg. This was epitheliomatous in type and apparently of stomodeal origin, extending from the sellar space where it had produced marked atrophy of the hypophysis, particularly the posterior portion, although there was determinable no evidence of actual neoplastic invasion of this tissue, a feature which, in the authors' opinion, affords explanation of the definite absence, in this case, of the so-termed hypophyseal syndrome.

RAPHAEL, Ann Arbor, Mich.

PERIPHERAL NEURITIS FOLLOWING MALARIA. JANUSCH, Ztschr. f. d. ges. Neurol. u. Psychol. **63**:123, 1921.

Janusch says that infection and intoxication are, of course, the principal factors in the causation of malarial peripheral neuritis, but that predisposing causes are exertion, exposure and undernutrition. Five personal cases are reported, occurring in soldiers in the Balkan campaign. The neuritis begins soon after the onset of the malaria, usually within two weeks. It affects the proximal groups of muscles of the extremities, and chiefly the shoulder muscles. In many cases the neuritis is limited to a single nerve, or even a single muscle. The occurrence of the disease in the muscles of the shoulder girdle has been laid to the unusual activity of these muscles brought about by carrying packs during the campaign. Paralyses are predominantly unilateral. Objective disturbances of sensation are uncommon, but there are often severe pains in the areas affected. There is a tendency to spontaneous recovery, but this is very slow, usually requiring about a year. The treatment is symptomatic and electrical. Quinin although curing the malaria, has no effect on the neuritis.

FREEMAN, Philadelphia.

PSEUDO-SYNDROME OF TAPIA. A. DE CASTRO, Rev. Neurol. **27**:537 (June) 1920.

The author's case was that of a tabetic patient (a woman, aged 35) in whom manifestations of eleventh and twelfth cranial nerve palsies developed following self-inflicted laceration (suicidal) on the right side of the neck. Examination revealed sternocleidomastoid and trapezius palsies, a nasalized voice, right atrophy of the tongue with marked fissuring, tremor and deviation to the right on extension and bilateral paralysis of the adductors and abductors of the larynx. There were also evident signs of the tabetic series—typical lancinating pains in the legs, lost knee and ankle jerks, the Romberg sign, hypotonus of the lower extremities, ptosis of the left eyelid, right pupil of the Argyll Robertson type, and a positive Wassermann reaction.

De Castro comments on the resemblance of the findings determined in this case to those characteristic of the so-called Tapia syndrome (homolateral glosso-scapulo-laryngeal hemiplegia depending on eleventh and twelfth nerve disturbance). Of definite interest in this case was the presence of bilateral laryngoplegia, a distinct departure from the conventional Tapian picture, which

(on the left) de Castro feels may well be syphilitic in basis and of central locus as opposed to the eleventh and twelfth nerve palsies on the right which seemed definitely peripheral in character, a feature particularly significant in view of the obscurity at present obtaining as regards the actual mechanisms involved in this syndrome.

RAPHAEL, Ann Arbor, Mich.

SPECIFIC TREATMENT OF DERMATITIS VENENATA (RHUS TOXICODENDRON). (PRELIMINARY REPORT.) ARTHUR SAYER, Med. Rec. **100**:717 (Oct.) 1921.

Eighteen case reports are given showing the results of treatment with tincture of *Rhus toxicodendron*. The tincture was prepared from the fluid extract as follows: fluid extract 2 parts, alcohol (95 per cent.) 9 parts, water 5 parts. An average dose of 12 minims was injected into the gluteal muscles. Twenty-five per cent. of cases showed immediate striking improvement; 25 per cent. more improved with one injection and were entirely relieved by a second injection. At least 75 per cent. of the cases were materially benefited. No local treatment was used in this series, but the author recommends the following to be used in conjunction with the injections: soap and water scrubbing at the onset, followed by an application of 1:1,000 potassium permanganate solution for five minutes, then calamine and zinc lotion every two or three hours.

TOMLINSON, Omaha.

ON THE PERMEABILITY OF THE CHOROID PLEXUS. A. BARBE, Rev. Neurol. **27**:314 (April) 1920.

Barbe carried out a series of carefully controlled perfusion experiments on the choroid plexi in a number of human subjects of various ages who had succumbed to various systemic and neuropsychiatric disorders. On the basis of this study the choroid was found to be most permeable to blood serum. Penetration of test fluids was noted to begin a few seconds following application, depending primarily on the freshness of the choroid material, the age of the subjects (seniles least permeable) and the absence of neuraxial disease. The actual rate of penetration through the choroid averaged approximately 1 c.c. per minute in normal subjects, remaining so for about twenty-four hours, after which alteration occurred as a result of postmortem changes. In certain conditions, notably general paresis (a finding of considerable therapeutic importance, if actually established) choroid permeability was found to be definitely increased, while in others, as in epilepsy, it was noted to be diminished.

RAPHAEL, Ann Arbor, Mich.

Society Transactions

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JAMES B. AYER, M.D., *President, in the Chair*

THE SOMATIC ORIGIN OF CERTAIN SEXUAL DELUSIONS. DR. J. W. COURTNEY.

Probably there is no delusional state more curious than the belief entertained by women that beings either human or supernatural have sexual intercourse with them during their sleep. According to the tenets of the oneirologic school of psychology, a delusion of this character is the product of an uneven conflict waged in the unconscious mind between erotic desires and moral inhibitions. The concept of it here propounded is based on fundamental physiologic principles—hence excludes the unscientific notion of an unconscious mind, since, obviously, mind cannot exist without some degree of consciousness.

When we trace to their origin those sensations whose summation is the sexual orgasm, we find that they are manifold and of several orders; tactual, muscular, visual, auditory, olfactory, thermal and so forth. As a result of the repetition of sexual congress at intervals over a certain period of time, these sensational elements become so strongly integrated and correlated that their resultant ideational and emotional complexes may easily be reproduced by stimuli, however indirectly applied, which evoke but a single one of the sensory elements enumerated—and that in so minor a degree of intensity that in its course along centripetal paths, it fails to reach the higher levels of consciousness.

With these physiologic facts in mind, the genesis of the delusion under consideration is manifest. In the sleep state tactual and thermal sensations initiated in the external genitalia by bed clothing, thermal sensations in the same parts aroused by the pelvic congestion of vasomotor origin which precedes menstruation, or the sensorimotor impulses sent direct to the clitoris by a full bladder through the medium of the pudic nerve, easily suffice to set in action the remainder of the group of integrated and correlated sensations, and these, according to the strength of their integration and correlation, determine the vividness of the ideational feeling of sexual congress to which, by sublimation in centers higher up, but still below the highest levels of consciousness, they give rise.

The concordance between the principles thus outlined and observed clinical phenomena is perfect. Where the deluded individual's experience with sexual congress is extensive, the dream coitus has an ideational content which embraces highly complex combinations—postural and dynamic—in the voluntary and involuntary muscular systems alike, intense tactual and thermal sensations and last, but not least, visual impressions of an order sufficiently vivid to give rise to the actual sight of a male figure, yet falling short, as a rule, of the degree of vividness necessary to identify this figure with that of a known person.

When the deluded person is virgo intacta and without personal sexual experience of any order or degree—when her knowledge of the male configuration has been derived through effigies, and of the procreative act by reading and hearsay, the sensational elements underlying her delusion are so feebly integrated and correlated that the story she tells of her defloration during sleep lacks utterly in circumstantiality and congruity.

In both instances, however, the ideas initiated by the sensational stimulus follow well-established physiologic laws as regards durability. Their tendency to endure is, to be sure, enhanced by the general erethism of the organism, which coexists. Be this as it may, they do in fact persist with such tenacity that the reasoning powers of the person's fully awakened consciousness are impotent to repress them. By their very persistence they soon dominate the mind as completely as if they were established facts—hence, readily excite its inferential activities.

In the case of the woman with actual experience of coitus these activities seldom go further than to lead her to the conclusion that the partner of her nocturnal sexual activities is a human being, whereas, in the case of virgo intacta without any personal knowledge of the procreative act, the conclusion arrived at is infinitely more complex. For the reasons already set forth, she cannot identify her ravisher even as a member of the human species—hence, through the religious exaltation of which she is generally also the victim, her distorted reasoning runs on supernatural agency, and she finally convinces herself not only that her dream defloration was brought about by such an agency, but that its accomplishment is of vast importance to the celestial hierarchy.

DISCUSSION

DR. STANLEY COBB: If it were true that such dreams had a physiologic rather than a psychogenic origin, all women at certain times would have such dreams. There must be a psychogenic factor and a subconscious or unconscious cause aside from the physiologic. The physiologic and psychologic theories are not mutually exclusive.

DR. J. W. COURTNEY: I do not consider the two theories mutually exclusive. I simply emphasize the physiologic as the dependable one, the one dictated by common sense. To exploit theories of pathogenesis in mental disorders, which in nowise conform to the laws underlying pathogenesis in general, seems to me highly illogical.

A PRELIMINARY REPORT ON THE TREATMENT OF PARALYSIS AGITANS. DR. HUGO MELLA.

Claude, Sicard, L'Hermitte, Quesnel and Rodriquez have reported excellent results with the use of sodium cacodylate in cases of spastic paraplegia and Parkinson's disease.

Stanley Cobb, at the Massachusetts General Hospital, gave sodium cacodylate in 0.5 gm. (7.71 grains) doses (intramuscularly) daily for ten days with no relief in two cases: one of paralysis agitans and one of amyotrophic lateral sclerosis. One patient with paralysis agitans showing marked tremor and moderate rigidity with pain had relief from the rigidity and pain, and the tremor diminished. At the Massachusetts General Hospital I gave it in one case of syphilis of the central nervous system with Parkinson's syndrome, and no amelioration of the tremors or rigidity was noted. One patient with rigid postencephalitic Parkinson's disease, after ten doses of 0.5 gm. (7.71 grains) and four of 1 gm. (15.43 grains) showed only slight relief from rigidity.

Another patient with a mild case of Parkinson's disease, after ten 0.5 gm. doses walked better, wrote better and could move her arms more freely. The results in ambulatory cases not being satisfactory, four advanced cases at the Long Island Hospital, Boston, were then chosen.

Case 1.—In this case rigidity commenced eighteen years ago. The patient was bedridden for the last year. She had tremor of the arms and legs which were markedly flexed and rigid. She could not feed herself or raise her arms. After the fourth dose of sodium cacodylate the patient could raise the right arm and feed herself.

Case 2.—Rigidity and tremors commenced twelve years ago. The patient was very rigid and could not raise his arms to the level of his shoulders. In April, 1921, ten doses of sodium cacodylate 0.5 gm. (7.71 grains) each were given with no relief. Then six doses of 1 gm. (15.43 grains) each were given after which the patient could raise his arms above his head, move more easily and showed only slight rigidity; but associated movements in rising from a chair and walking did not return. He has received no injections since that time and his condition is about the same as when treatment was discontinued.

Case 3.—The patient had been practically helpless for the last four years; she had to be lifted from the bed to the chair and could not feed herself; she had marked tremors and rigidity. After fifteen 0.5 gm. doses of sodium cacodylate in April, 1921, the patient could walk across the ward unaided, raise her arms above her head and feed herself. One course of ten doses has been given since then and the patient is still up and feeds herself.

Case 4.—Rigidity and tremors commenced four years ago. The patient had been confined to bed for eighteen months, practically helpless. In April, 1921, after eleven 0.5 gm. doses of sodium cacodylate, the patient was up, walking about the ward and able to feed herself, showing a slight tremor and only a moderate amount of rigidity.

A PRELIMINARY REPORT ON THE TREATMENT OF MYASTHENIA GRAVIS. Dr. Hugo Mella.

In the fall of 1920 a young lady was seen in the outpatient department of the Massachusetts General Hospital who showed the signs and gave the typical electrical reactions of well-developed and advanced myasthenia gravis. Just previous to this a patient under my care had died of this disease and necropsy showed a large, malignant, thymus gland. A roentgenogram of the case showed a rather large shadow in the thymus region. Roentgen-ray treatment was commenced by Dr. Holmes, under great precaution, in suberythema doses (the patient reacting severely). After seven such doses the patient was entirely relieved. She has been free of all signs of myasthenia gravis since April, 1921. Her electrical reactions are now normal. A complete report of this case is to be given when at least a year has elapsed after the disappearance of all signs of the disease.

DISCUSSION

Dr. Stanley Cobb: Our interest in these problems has been aroused by the work of the French authors mentioned. While the experience at the Massachusetts General Hospital has been somewhat unsatisfactory, those cases were all ambulatory cases. The most marked improvement has been in the cases at the Long Island Hospital in which there was often extreme rigidity of long standing. It seems certain that arsenic affects the rigidity of paralysis agitans in some way. Recent investigation in the Department of Neuro-

pathology at the Harvard Medical School so far corresponds with the recent work of Wilson and Ramsay Hunt; that is, in all probability the pathology of rigidity and tremor lies in the basal ganglions.

DR. A. S. MERRILL: The diagnosis of enlarged thymus is not an easy one to make. Occasionally a shadow can be seen which can be attributed to nothing else, but the thymus generally is transparent and casts only a slight shadow. The thymus is known to be one of the most susceptible organs in the body to the effects of irradiation. Assuming that the patient's trouble is due to thymus hyperactivity, it would be quite logical to presume that irradiation of this gland would have some effect on the patient's condition.

DR. WALTER B. SWIFT: A year or two ago I proposed a series of exercises for the treatment of paralysis agitans. At that time I could not exactly estimate their value, but from fifteen or twenty additional cases I judge the improvement to be elimination of from 10 to 60 per cent. of the tremor. When a man can be made to hold his newspaper still so that he can read it, and when he does not require an hour or two to go to sleep, it would seem that the exercises are of some help. They are not a cure, and some cases do not respond. The trouble seems due physiologically to some lack of conscious sensorial control.

DR. J. W. COURTNEY: In two necropsies of paralysis agitans cases made a number of years ago, the most striking thing observed was the apparent shrinkage of the cord in toto—apparently not over half its normal size. In one of these cases Dr. Thomas made serial sections through the entire cord and about all that was found were universal perivascular changes of no marked extent.

DR. MELLA, closing: There is a great difference of opinion as to whether the lesion of paralysis agitans is located in the globus pallidus (Ramsay Hunt) or in the locus niger (Trétiakoff). There are several structures sending impulses to the red nucleus, of which even the physiology is not clearly understood, much less the pathology.

The intravenous method could be used, but the intramuscular injection had been found to be simpler. Furthermore, the arsenic preparations are excreted rapidly when given intravenously, but much more slowly when given intramuscularly. It is also less painful than if given subcutaneously. Aseptic precautions are necessary.

A CASE OF SYRINGOMYELIA TREATED BY THE ROENTGEN RAY.

DR. GEORGE CLYMER.

In 1907, a series of cases of syringomyelia, treated by the roentgen ray, were reported by Beaugard and L'Hermitte. Their report was so encouraging that further observation seemed desirable. The present patient, a young woman, aged 24, single, has been under observation for a little over six years. The duration of the symptoms, when the patient was first seen, was about eighteen months. There was a history of weakness and atrophy of the right hand, disturbance of sensation, chiefly of heat and cold, and pains running from the spine into the right arm. Examination revealed an area of diminished sensation involving the inner aspect of the right arm and hand, extending to the back and chest, and also an area in the right thigh. A roentgenogram of the spine revealed bifurcation of the spines of the third and fourth cervical vertebrae, suggesting congenital developmental defect. Under roentgen-ray treatment the patient's symptoms have grown less, the pains have ceased, and she has

held a secretarial position for three years. A recent careful examination revealed diminution in the area of sensory disturbance. There was no evidence of progress of the disease.

DISCUSSION

DR. A. S. MERRILL: The effect of the roentgen ray on cell tissue depends on its approach to the embryonic form, that is, cells approaching the embryonic type with active cell division are more susceptible to the roentgen ray. Assuming that this condition may be due to proliferation of neoplastic cells approaching the embryonic type, it is reasonable that they may be affected by the roentgen ray provided they can be reached by a proper dose. The patient of Dr. Clymer, on that theory, received treatment over the whole cervical and practically the whole dorsal cord. The irradiation was pushed to the limit of skin endurance. French authors are very optimistic in their statements. However, eleven months was the longest time a patient was followed. Dr. Clymer's patient had been under observation for over six years. Improvement began promptly after the first few applications, motor symptoms improving first. As might be reasonably assumed, there is a symptomatic residue probably due to destruction of certain nerve tracts. Several cases of fair duration have showed encouraging results. Dr. Clymer's patient was irradiated thoroughly over sixteen or seventeen areas from the upper cervical to the lower dorsal cord on either side. At that time the method of cross firing was used to pick out the objective point from different directions and get a multiple dose at the spot where the growth focused and also to avoid injuring the skin. The French authors state with perfect reason that the cord should be treated over the whole extent indicated by the most careful neurologic examination and, it may be added, then some. Another patient under treatment has given less encouragement. So far as can be seen, there is little change in the patient's condition. She has received a number of irradiations including only the cervical cord. It would be better if she could have further irradiation over more of the cord.

DR. J. B. AYER: A woman aged 27 was given roentgen-ray treatment beginning in 1919. She had a very weak, atrophic left hand and much difficulty in distinguishing heat and cold. Four months previous to her coming she had developed distinct weakness of the right hand. The history, therefore, indicated progression. The blood and spinal fluid Wassermann reaction were negative. She became worse in 1919 and developed paralysis of the left vocal cord. She was then given roentgen-ray treatments. From December, 1919, to June, 1921, she was given seventeen roentgen-ray exposures by Dr. Merrill. She showed no progression in that time. On the contrary, she felt much better. It seems possible that this case has been arrested although not much improved.

DR. CLYMER, closing: This is, at least, a form of treatment worth considering.

RESULTS OF A BRIEF NEUROPSYCHIATRIC EXAMINATION OF ONE THOUSAND ONE HUNDRED AND FORTY-ONE STUDENTS. DR. STANLEY COBB.

In 1914 Dr. Roger I. Lee began the physical examination of students entering Harvard. The examination was necessarily brief; later he called in certain specialists to assist. He asked me to study "nervous instability" because of the common occurrence of neurotic symptoms. The results and the method of working on the problem follow:

First we picked out men who could give a past history of some neurotic condition; second, those who presented physical signs indicating a tendency to nervous instability; third, those who showed evidence of endocrinopathy. Then we followed up through the four years the men on whom primary observations had been made, in order to study their reactions to their problems. The correlation of these findings showed first the incidence of nervous instability. Eighty-eight of the 1,141 had a definite past history of some form of nervous instability, such as night terrors, sleep walking, stammering. This last trouble comprised 23 per cent. of the neurotic symptoms. A comparison was made of the physical findings in these eighty-eight men with those in men who had no such history. The pulse rate, vasomotor symptoms, knee reflex, posture, urine, blood pressure and the vegetative nervous system were studied. Of the 1,141 students only twenty-two showed a pathologic condition of the glands of internal secretion, and they did not have severe cases.

The work indicated these conclusions:

1. History is the best guide to nervous instability—family history, past history and present complaints.

2. In a fifteen minute examination it is impossible to obtain an accurate history, and freshmen have few well-defined problems.

3. Vasomotor instability was found somewhat more frequently in men with neurotic histories.

4. Tachycardia, blood pressure variation and dermographia are often found associated with each other and with exaggerated knee reflexes. Men with albuminuria are likely to show all these symptoms.

5. Endocrinopathy was rare, but the small number of cases discovered showed more symptoms referable to the vegetative nervous system and less neurotic history and acne.

6. In men with poor mechanical use of the body, tachycardia, sinus arrhythmia, high blood pressure and variable systolic pressure were more common.

7. The men with poor bodily mechanics passed better psychologic examinations than those with poor posture.

DISCUSSION

DR. LLOYD T. BROWN: The examinations were carried out at the end of the vacation when the men were in the pink of physical condition. In the literature it was possible to find no standard by which to judge what constituted good and bad bodily mechanics. Therefore, tracings were made of each man, and according to the appearance of these tracings four groups were made. About 740 men were examined.

In judging the posture, or bodily mechanics, four points were taken up: the position of the head, the position and shape of the chest and abdomen, their relation to each other, and lastly, the anteroposterior curves of the back. It was found that 7.5 per cent. fell in the A, or good bodily mechanics group; 12.5 per cent. in the B, or fairly good group; 55 per cent. in the bad bodily mechanics group, and 25 per cent. in the very bad bodily mechanics group. These men were all asked about the occurrence of backache. No man in the A or B groups had backaches; about 7 per cent. of the C group and 9 or 10 per cent. of the D group had had backache. Operation for appendicitis had been performed one and a half times as often in the C and D groups as in the A and B. Albuminuria was found in a considerable number of men. These were followed up with great care; seven of them had persistent or orthostatic albuminuria. Of these seven, one was in the C, the other six in the D group.

In judging the postures or the mechanics of the body, it is necessary to appreciate that there are very different types of individuals and no one standard for the human race as a whole. Some people are naturally thin with long, thin bodies and will never grow fat. Others are short, stocky and tend to grow fat. Either of these two classes or the very large class that is half way between, may use their bodies very badly mechanically, so that it is necessary to judge all people from a standard which can apply to one particular person.

Bad bodily mechanics are common in children, associated with fatigue. For instance, a child 4½ years old, always a difficult child to feed, who was never really well, having many attacks of indigestion and constipation, was brought to an orthopedic surgeon because of acute attacks of extreme pain in one hip. Examination showed no organic disease of the hip, but the child used her body in an extremely faulty mechanical way. She was shown how to use her body properly and given a brace to relieve the fatigue. She recovered entirely from the hip trouble and the constipation and indigestion were improved. After two years she came back with the original symptoms. The postural work had been entirely given up. It was again begun under more strict supervision and at the present time the child is 16 years old and is well.

Such a case shows that it is necessary to recognize the faulty mechanical element as well as the fatigue element, and that the remedy for these conditions is education not only of the child, but of the parents; also that this education should be started when the child is very young. It should be a part of the curriculums of all schools, so that when adult life is reached the necessity for the individual to compensate for his poor bodily mechanics will not add one more element of fatigue.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 28, 1921

GEORGE WILSON, M.D., *President*

A PATIENT WITH MYELITIS AND NEURITIS. Presented by DR. S. F. GILPIN.

The patient, an Italian woman, aged 31 years, was admitted to the Jefferson Hospital, April 16, 1921. Her chief complaint was loss of sensation and motion in the lower extremities. Her health had always been good and the past medical history was negative. She was married and had four children living and in good health and had had one miscarriage.

Her present trouble began April 1, 1921, with a sensation of coldness and numbness in the right foot. This improved slightly, then paresthesia started in the left foot and extended up to the waist line, and at the same time the right leg became involved in its entirety; all this occurred within one week. She then began to feel weak in the left leg and limped about for one week; then the left leg became entirely helpless and the right limb weak. Two weeks before admission she went to bed and in a few days both legs became helpless. She had no headache, did not vomit and was not dizzy; there was no impairment of vision and no pain. She had loss of control of both sphincters for ten days previous to admission.

Physical examination showed that the patient was well nourished and mentally clear. The pupils reacted normally to light and in accommodation; the cranial nerves were normal and the general examination was negative. The patellar reflexes were absent; there was no ankle clonus, but Babinski's sign was present on both sides. She was hyperesthetic to pressure over the shoulders, upper part of the chest, lower extremities and abdomen. There was complete loss of motion in the legs. An ulcer was present on the left thigh. Sensation was lost in both legs, and the patient complained of fulness in the abdomen, suggesting a girdle sensation. A roentgenogram of the spine showed no pathologic condition. Examination of blood and urine was negative, as was the Wassermann test of the blood. The spinal fluid was normal. The patient gradually improved so that in six months she was able to walk with support. The deep reflexes returned, the superficial reflexes were normal, and she regained perfect control of the bowels and bladder.

The diagnosis of acute transverse myelitis seemed to be correct, but she developed signs of multiple neuritis. Did she have both myelitis and neuritis? Can a patient with such marked symptoms of a transverse cord lesion recover, or has she a case of neuritis with unusual involvement of the sphincters? The patient appears to be recovering with no signs of a permanent cord lesion.

DISCUSSION

Dr. Charles K. Mills: The history of this case is suggestive of a neuromyelitis, from which the patient may largely recover, that is, from which she may make approximate recovery. It did not seem to have been a syphilitic case, although its origin was evidently due to an infection.

Dr. Francis X. Dercum: I regard the case as one primarily of transverse myelitis in which a multiple neuritis was present. I have nothing to add to the description given by Dr. Gilpin, with the exception of emphasizing the sensation described by the patient of a broad constricting band around the abdomen. The clinical picture was unusual, and the patient made an unusual recovery.

A CASE OF SYRINGOMYELIA. Presented by Dr. S. F. Gilpin.

The patient, a Russian miner, was admitted to the Jefferson Hospital, Sept. 14, 1921, complaining of loss of power and of motion in the right arm. The personal and the past medical history were negative.

Present Illness: Two months before observation he noticed a gradual weakening of his right shoulder and upper part of the arm. The arm felt heavy and weak, but he had no pain.

Physical Examination: He could not raise the right elbow above the level of his shoulder. The grip in the right hand was normal. The eyes, chest and abdomen were normal. The spine showed marked scoliosis. The patellar reflexes were increased, but there was no Babinski or clonus. The reflexes were diminished in the upper extremities. The muscles of the right shoulder girdle were atrophic. Sensation was normal except over the region of the shoulder girdle. In this area he was unable to differentiate between a blunt and a sharp object and did not recognize heat and cold. The blood Wassermann test and test of the urine were normal. The roentgen ray showed right lateral scoliosis of the dorsal vertebrae with a moderate amount of osteo-arthritic changes involving virtually all the dorsal vertebrae. The patient appeared to have a case of syringomyelia with fibrillary tremors in the atrophied muscles, and the large muscles of the shoulder girdle were the first muscles to show atrophy.

DISCUSSION

DR. F. X. DERCUM: This condition began, as far as could be determined, in the hand, as is commonly found in beginning syringomyelia. The interesting feature is that the sensory loss of pain and temperature follows the shoulder girdle where the wasting is at present most marked.

DR. CHARLES S. POTTS: It is worth considering whether the symptoms are not due to the so-called spondylose rhizomelique, which is nothing more than an arthritis of the vertebral joints. In the so-called von Bechterew type there are symptoms of cord disturbance, especially of the nerve roots, which might cause the symptoms present in this case. It would seem to be especially so in view of the fact that there are no evidences of involvement of the spinal cord itself.

DR. WILLIAM G. SPILLER: I recently had a patient with syringomyelia of the scapulohumeral type in my service at the University Hospital. This type is uncommon. It is possible from the hypertrophic arthritis of the vertebral column mentioned by Dr. Gilpin that his case is one of pachymeningitis cervicalis hypertrophica of Charcot and Joffroy, which sometimes clinically resembles syringomyelia.

DR. JOHN H. W. RHEIN: In 1908 I made a pathologic report of the nervous system in a case of spondylose rhizomelique in which there was degeneration of the spinal roots. I believe that the symptoms present in Dr. Gilpin's patient might be explained on the basis of degeneration of the roots as a result of pressure of the diseased bones of the spinal column.

A PATIENT WITH DYSTONIA MUSCULORUM DEFORMANS. Presented by DR. N. S. YAWGER.

The case was that of a man of Irish ancestry, 21 years of age, who appeared to have had some defect since birth. He was an eight months' baby; the labor was induced, difficult and instrumental. His weight at birth was 5 pounds, and for years he was unhealthy and always had peculiar movements. His teeth did not erupt until the seventh year; he did not creep until 14, and although since that time he has shown much improvement in his ability to get about, he has never been able to perform such definite movements as feeding himself. It is said that he could not hold his head erect until he was 10 years of age. Of recent years, except for constant, spontaneous movements, he has been fairly healthy and eats ravenously.

The family history was without interest except for the fact that the mother was only 4 feet and 7 inches in height and a maternal aunt was of the same stature. The mother had had nine pregnancies, three of which resulted in miscarriages; on account of her small pelvis, labor had always to be induced at the eighth month.

Physical examination revealed physical stigmas shown by the palatal arch which was narrow and shallow, by teeth that were prominent and by a converging insufficiency of the right eye. The patient could get to the erect position and stand unsupported for a few seconds. Attempts to walk caused a more or less violent struggle of his musculature. He could not walk alone for more than a half dozen steps, during which time he inclined backward and often fell. If he walked for any distance he followed along the side of a room, or he had to be strongly supported by at least one person. His indoor mode of progression was preferably on his hands and knees, and was accomplished through a series of almost springing movements. Most of his movements were made suddenly and when he attempted to do anything he appeared

to take his muscles by surprise. The patient had a three-wheeled conveyance which he worked with his feet and guided with his hands, and though it seems almost incredible, he went about alone. The choreiform movements almost never ceased except when he was sleeping soundly; even the muscles of the head and face were involved. The muscle power was a little weak throughout and there was marked incoordination. The tendon reflexes were normal in the upper extremities but were slightly exaggerated at the knees and the ankles. Ankle clonus and the Babinski sign were absent. The pupils responded normally to light; there were no sensory changes or definite atrophy and no paralyses or sphincter involvement. The speech was hesitant, explosive, difficult to understand, and there were momentary periods when he could not speak at all. The patient had no gross mental defect. He was illiterate, but his memory was good. He was able to make his family understand him and was acutely aware of all that was going on about him. The blood Wassermann reaction was negative.

Much interest attaches to the part of the brain implicated in this defect. The disorder has been styled dystonia lenticularis. It has been placed in the group of corpus striatum diseases. Dr. J. Ramsey Hunt, writing recently, reconstructs the corpus striatum on histologic and functional lines, by dividing it into neostriatum (caudate nucleus and putamen) and paleostriatum (globus pallidus). To the former he assigns a coordinating and an inhibitory influence on the motor functions of the corpus striatum. Involvement here, leads to choreiform or spontaneous movements of the automatic associated type. Hunt regards dystonia musculorum deformans as related to the neostriatum.

DISCUSSION

DR. N. W. WINKELMAN: Two or three things are suggested in this discussion. In studying the basal ganglions pathologically one is struck by the similarity of the caudate and putamen. Both are identical histologically, and they are developed at the same time and from the same structure. The globus pallidus is developed at a totally different time and has a totally different structure. The lenticular and the caudate nucleus have the same type of cell, a motor type such as is found in the cerebral cortex in the motor area and in the anterior horn cells. In certain conditions, especially in paralysis agitans, there is a disappearance of the large motor cells from the basal ganglion not limited to the globus pallidus, while in Huntington's chorea the pathologic condition is found in the smaller type of cells. There is no sharp anatomic delimitation of these two types of cells, for while the corpus striatum is composed mainly of the smaller type of cell it still contains some of the larger motor type of cells.

DR. WILLIAM G. SPILLER: There is a striking relation between some of these disorders of the lenticular nucleus. It is interesting but not contradictory that certain members of a family have this disorder, because I believe there is a close connection between dystonia lenticularis manifestations. I have been in doubt whether the functions of the different parts of the lenticular nucleus can be definitely separated from one another; whether there can be destruction of the globus pallidus without organic or at least functional disturbance of the putamen.

In the case of double athetosis with bilateral destruction of the putamen, which I reported in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, volume 4, October, 1920, the destruction of the putamen was as nearly isolated as one can expect, and yet the globus pallidus was not normal.

DR. CHARLES K. MILLS: I have always been interested in this class of cases, particularly because I have expounded before this Society more than once my belief in a separate extrapyramidal system, which I sometimes call the tonectic cerebral apparatus.

DR. F. X. DERCUM: I agree in general with Dr. Mills as to the interpretation of cases of this kind. Possibly there are two factors to be considered. Dr. Yawger's case was that of a patient born at eight months, and in all probability with a failure of normal cortical motor development; the axons of the cortical motor neurons had probably never fully completed their course to the cord. The normal tonicity of the muscles is due to the interplay of the motor cortex and of the subcortical motor centers, especially the globus pallidus. If one or the other, that is, either the pyramidal or the pallidal system be diseased, disturbances of muscle tonus must make their appearance. The normal balance between motor impulses descending from the pyramidal tract and those descending from the pallidal system are no longer counter-balanced and are no longer mutually inhibited, and one being destroyed or imperfectly developed, we have an overflow over the other. It is the pyramidal overflow which gives rise to the spasticity and rigidity in paralysis agitans; it is the pallidal overflow which gives rise to the rgidity in hemiplegia. In the present case the deficiency of development of the motor area of the cortex throws the balance over the subcortical motor centers, the corpus striatum. Hunt believes that the disorders of association should be referred to the caudate nucleus, a position which, together with others, I am strongly inclined to accept. In the present instance the symptoms are obviously to be referred to both caudate nucleus and the putamen on the one hand and the pallidum on the other. We should bear in mind, also, the facts of morphology. In reptiles and birds the striatum is the cerebral motor organ. In mammals, it is superseded and its function usurped and inhibited by the pallium, the cortex, which appears so much later in the course of evolution. We can perhaps understand, when the motor area of the cortex fails of full development, that the striatum should be left to act unrestrained and uninhibited; or on the other hand, the striatum having failed of development or undergone disease, we can understand that there should be both spasticity and absence of association of movements. Probably the present instance is complicated by the fact that both the motor area of the cortex and the striatum are each in some degree impaired; probably the striatum has suffered most.

DR. CHARLES S. POTTS: The case shown does not seem to present the typical symptoms of dystonia musculorum. There is not present the violent spasm of the muscles about the pelvis, the torsion movement, which has given one of the names, tortipelvis, sometimes applied to this condition. Lordosis is absent, and in the other members of the family afflicted with the disease there had been marked mental deterioration which is not a symptom of dystonia. It seems to me that as dystonia musculorum and Huntington's chorea are both believed to be due to disease of the lenticular region, a good deal of this question might be distinction without a difference.

DR. CHARLES M. BYRNES: I am quite certain that Dr. Ramsey Hunt has made a distinction between symptoms due to a lesion in the globus pallidus and those associated with a lesion in the putamen. I agree with the opinion expressed by Dr. Spiller that such a distinction is, perhaps, not clinically possible.

In connection with the extrapyramidal system, I wish to relate an observation suggested by the study of Sinkler's toe reflex presented to this Society

by Dr. Spiller. I repeated the experiment on a case of spastic paraplegia in which it was observed that the Babinski toe reflex, the ankle clonus and the spasticity were abolished for a period of five minutes after the release of the flexed toes. The same experiment was tried on a case of old hemiplegia in which, however, flexion of the toes failed to relieve the spasticity or alter the reflexes. I believe that in the latter instance, failure of the reflex action might be due to preservation of extrapyramidal function.

A CASE OF EPIDEMIC ENCEPHALITIS PRESENTING UNUSUAL SEQUELAE. Presented by DR. GEORGE E. PRICE.

In January, 1920, a boy, aged 11, became ill with epidemic encephalitis, presenting fever, diplopia, somnolence, etc., and had apparently recovered when in the following July he developed attacks of dyspnea. He complained of headache and dizziness; breathing would be suspended for a brief interval, when he would become cyanotic; then would follow a period of rapid breathing. One such attack, timed by Dr. Sellars, lasted for two and one-half minutes. The child would have no recollection after the attack as to what had occurred. While remaining about the same in frequency, an average of four or five attacks in twenty-four hours, the spells gradually changed somewhat in character, losing the earlier period of suspended respiration and cyanosis.

When examined in September he was having paroxysms of very rapid, difficult breathing and markedly accelerated heart action, accompanied after from thirty to sixty seconds by an intense frontal headache. The child retained consciousness throughout the attack to such an extent that he would respond correctly when spoken to, yet after the spell was over he would have no recollection of what he had said. It was noted that the paroxysms occurred most commonly in the morning, shortly after waking. A careful neurologic examination was negative; the heart and lungs were normal and the urine was negative.

The child's history previous to the attack of encephalitis was uneventful from a medical standpoint, and the family history had no bearing on the case.

The only plausible explanation for this syndrome would seem to be that there is an intermittent disturbance of the centers in the medulla, of vascular origin, occurring as a sequel of the encephalitis. These so-called "bulbar crises," while rare, have been observed by other writers.

THE SYNDROME OF THE LONG FIBERS OF THE CROSSED PYRAMIDAL TRACT (SUBACUTE DEGENERATION). Presented by DR. ALFRED GORDON.

After a historical review of the subject of subacute degeneration of the white columns in the spinal cord, the speaker called attention to cases of posterolateral sclerosis without anemia. A differential diagnosis between the classic combined sclerosis and that occurring from subacute degeneration of the posterior and lateral columns of the cord was emphasized. Dr. Gordon then discussed the pure type of lateral sclerosis, of which he found only six cases in the literature. He took up the main subject of his observations, namely, the possible occurrence of a subacute degeneration confined exclusively to the pyramidal tract in the cord. He had under his observation for several years two patients whose clinical picture was not at all that of primary lateral sclerosis, but nevertheless there were striking evidences of involvement of the motor tract in the cord. At no time was there a rigidity of the affected

limbs, but the patellar tendon reflexes were markedly exaggerated, the plantar reflexes were extensor in type, and ankle clonus was present. There were no sensory disturbances and no sphincter involvement. Spasmodic involuntary contractions of the muscles of the affected limbs were present throughout the illness. Dr. Gordon believed that besides the classical posterior and posterolateral sclerosis there exists a subacute involvement of the posterior, of the posterolateral and finally of the sole lateral (motor) tracts.

CHARCOT JOINTS OF THE SPINAL VERTEBRAE. Presented by Dr. F. X. Dercum.

Dr. Dercum exhibited the roentgenograms of two cases of Charcot spine. In both instances the patients suffered from undoubted and typical tabes; in one of them there was in addition a large arthropathy of the right knee; also exhibited in a roentgenogram.

Dr. Dercum said that he wished to emphasize the differential diagnosis between Charcot spine and tuberculous spines. He especially dwelt on the significance of the absence of fixation, the absence of muscular spasm, of mobility, and especially the absence of pain. The roentgenograms of Charcot and tuberculous spines may superficially resemble each other. He thought they should not be interpreted from roentgenograms alone, but from the clinical signs as well. By themselves the reontgenograms may unquestionably lead to error.

Book Reviews

KORPERBAU UND CHARAKTER (PHYSICAL DEVELOPMENT AND CHARACTER). By Dr. Ernst Kretschmer. Berlin: Julius Springer, 1921.

Dr. Kretschmer's researches contain a great many interesting and valuable suggestions, even though one may not be willing to accept all his conclusions. Briefly: He has discovered a more or less persistent line of parallel development between the physical characteristics of his mentally diseased patients and the types of mental disturbance which they present. It is not the old idea of the body influencing the general trend of the mind or of the mind stamping the body with certain definite stigmas. Stated in such a way the thesis of his book might seem vague and subjective, while, as a matter of fact, his method of procedure is intensely objective and accurate. The time was when we thought of human beings as divided into two compartments, "mind" and "body," and considered each compartment separately. "Mind" was enclosed in one air-tight compartment of the personality, while side by side with it was another compartment which we termed "the body and its functions." These two compartments were so clearly outlined that although they might have points or even surfaces of contact, there was no interaction between them. The psychiatrist when attempting to describe the influence of the mind on the body was forced to overcome a distrust lurking in the background of most people's thought—a well grounded unwillingness to believe in the existence of some mysterious mental power which might enable "the mind," in its compartment, to destroy the wall between it and "the body," and so to reach the physical functions by means of a break in its own continuity. Such a break suggested, at the least, an abnormal condition either of the body or the mind, or of both. Fortunately, this old idea of the two noncommunicating compartments, although it still persists in the mind of the laity—to a degree often unappreciated—has ceased to dominate medical thought and practice. In its place we have won our way to the conception of integration, to the functional unity of the personality, in which there is no dividing line between the physical and the mental. Viewed from this standpoint, Dr. Kretschmer's ideas are possible and acceptable.

He began his work by making very complete and detailed physical measurements and descriptions of the bodies of his patients. From a classification of these measurements several types of physical outline naturally emerge and become more or less clearly defined. Kretschmer distinguishes three principal types: (1) an asthenic type, (2) an athletic type, and (3) a pyknic type (from the Greek πυκνός = fat). There are a few other less numerous groups which do not fit exactly into any of these three major classes and which are composed of mixed types, which the author calls "dysplastic," "polyglandular," "infantile," or "hypoplastic." These last, however, are borderline cases which appear to Kretschmer to show symptoms of a disturbed endocrine balance.

Having thus first classified the physical characteristics of his patients, he has discovered that the athletic and the asthenic types are always associated with schizophrenic disturbances, while the pyknic type suffers from manic-depressive psychoses or, as he prefers to call it, from a "cycloid diathesis." His conclusions lead him also to a tentative theory of temperament. He finds that his "asthenic" and "athletic" physical types exhibit those mental

reactions which reach their extreme pathologic expression in cases of schizophrenia, while a pyknic physical habitus is always associated with a temperament that varies between the pathologic extremes of excitement and depression. The practical value of such a fact is self-evident. There are, for instance, certain evident hallmarks of the athletic and the asthenic type (for example, the comparative measurements between the circumference of the shoulders and that of the pelvis, or types of hair formation), which enable the psychiatrist to classify a patient who shows these characteristics as being likely to develop a schizophrenic psychosis rather than a manic-depressive one. This, is especially valuable in cases in which the psychosis is in its incipiency and in which it is possible to confuse a beginning schizophrenia with a mild depression. The reviewer has taken the trouble to test a number of Dr. Kretschmer's physical "sign posts" and in the brief period during this experiment has been greatly helped in the prognosis of puzzling cases.

It may be interesting to give a brief outline of the different physical types and their more important characteristics, which Kretschmer has outlined.

1. The asthenic schizophrenic type is distinguished especially by the small skull, the profile with its comparatively long nose, and the face by an outline resembling a shortened egg; more especially still by the acute angle of the profile, one side of which runs from the receding forehead downward with its apex at the point of the prominent nose and with its other side running from the apex of the nose downward and inward to the point of the receding chin. The body hair of this type is also characteristic. There is an excessive development of primary hair. The hair of the head is not only very thick, but it extends beyond the normal borderlines. It grows low over the occiput, and especially on the forehead and temples so that the usual bare spot at the temples is covered with fine hair; and there is a more or less definite bridge of primary hair between the terminal hair of the temples and the temporal ends of the eyebrows. The eyebrows themselves are very broad and thick and flow together over the base of the nose.

2. The athletic schizophrenic face has an outline that is egg-shaped, but that is lengthened instead of being shortened. The bony skull is larger, also more massive and higher. Especially characteristic are the broad outstanding shoulders, the deep chest, the firm receding abdomen which grows slighter as it descends so that the pelvis and the legs appear almost adolescent in type in comparison with the heavy arms and the hypertrophic shoulders.

3. The pyknic-cyclothymic type has also a very distinctive facial outline. It is flat and distinctly five cornered; the two temples, the two apexes of the angles of the lower jaw and the point of the chin forming the sides of the pentagram. The body is characterized by the excessive development of the thorax and abdomen in comparison with the rest of the body. The tendency to fleshiness is confined to the body, the extremities being much slighter and much less heavy. It is difficult to describe in a few words the characteristic differences of these types, which, however, spring to the eye at once when one analyzes the excellent photographs that are included in Kretschmer's book and which, unlike the photographs in many psychiatric monographs, form with the surrounding text an inherent unity of interest. There is not a photograph in the book that is unnecessary or mere padding.

Kretschmer, however, is not satisfied simply with presenting the facts of his thesis. He has a most fascinating chapter on "Die Genialen" (types of men of genius), in which he discusses the cyclothymic type of artistic tem-

perament, as well as the schizothymic type among artists, authors, scientists, philosophers and diplomats. The amount of general knowledge and reading that lies back of this single chapter is in itself enormous.

Kretschmer's book ends with a brief chapter on his "Theory of the Temperaments," which is in itself so suggestive, so full of new points of view that it deserves to be read carefully by every one who is interested not only in those stray types which drift as patients into his office, or his clinic, but who has a broader desire to understand the varying human elements with which his daily life brings him in contact; and even to get a still deeper insight into the reactions of those groups of persons who make up the composite pictures of different peoples and nations.

He constantly draws the line between what science knows and what he himself seeks to prove on the basis of such knowledge, between his scientific data and his personal conclusions. It is quite possible that the reader may not entirely agree with much that he has written. There are, of course, many gaps in his evidence which later research may be able to fill. On the whole, however, a book such as this, which bears on every page the stamp of patient investigation, is not only a definite contribution to our understanding of human personality, but is also an inspiration to thought and to further investigation on the part of every one who will read it carefully from beginning to end.

ACUTE EPIDEMIC ENCEPHALITIS (LETHARGIC ENCEPHALITIS). An Investigation by the Association for Research in Nervous and Mental Disease. Report of the Papers and Discussions at the Meeting of the Association, New York City, Dec. 28 and 29, 1920. Prepared under the direction of WALTER TIMME, M.D.; PEARCE BAILEY, M.D.; LEWELLYS F. BARKER, M.D.; SANGER BROWN, 2D, M.D.; CHARLES L. DANA, M.D.; J. RAMSEY HUNT, M.D.; FOSTER KENNEDY, M.D.; T. H. WEISENBURG, M.D.; GEORGE H. KIRBY, M.D.; HUGH T. PATRICK, M.D.; BERNARD SACHS, M.D.; WILLIAM G. SPILLER, M.D.; ISRAEL STRAUSS, M.D.; E. W. TAYLOR, M.D., and FREDERICK TILNEY, M.D. Price, $2.50. Pp. 258. New York: Paul B. Hoeber, 1921.

In December, 1920, the Association for Research in Nervous and Mental Diseases held its first meeting; two days were devoted to the study of lethargic encephalitis. There were thirty-five contributors, to each one of whom some branch or phase of the subject had been assigned by the Commission of the Association because of his known attainments. There was no general discussion of the papers presented, but the contributor was expected to defend his thesis, if necessary, and to answer all questions.

The total output of the conference was taken in hand by the Commission, sifted and arranged; duplications were eliminated and superfluous matter deleted. What was left was put into book form by the Publication Committee. The material is arranged in seven chapters and covers the subject from history, incidence and etiology through symptomatology (three chapters), diagnosis, prognosis, morbid anatomy and bacteriology to animal experimentation and immunology. The book certainly is the best thing on this subject that has appeared.

The Publication Committee very wisely has included "all questions of clinical bearing submitted by the Committee and answered by the contributors." Doubtless many of these questions would have occurred to the reader. He finds them answered.

Another feature of great value is the "conclusions of the Commission" which terminate each chapter. The Commission, in short, sits as a jury on

the communications and delivers its verdict. As the members of the Commission are nearly all known leaders of neurology in this country, their verdict may be considered about as final as any verdict can be in the present flux of medical knowledge. A remarkably full bibliography, eighteen pages, closes the volume.

That the profession and the public owe a great debt to every one concerned in the production of this volume will quickly be apparent to any reader.